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Docket ID No. EPA-HQ-OA-2018-0259

Comments submitted on the Docket Subject titled

Strengthening Transparency in Regulatory Science
Comment on Strengthening Transparency in Regulatory Science, Environmental Protection Agency, 40 CFR Part 30, RIN 2080—AA14 [EPA-HQ-OA-2018-0259; FRL-9977-40-ORD].

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The Submitter's opinions are personal and not attributable to the US Army or Department of Defense.

This submitter has witnessed US EPA misconduct for a period of 3 decades on a scale that is stunning, or alarming, going back to the EPA decision to ban DDT in the early 1970s, resulting in the deaths of millions in the 3rd world, and a particularly horrific impact on children.

More recently in addition to serial misconduct with regards to toxicology and epidemiology research the EPA has compounded its scientific methodology misconduct with a systematic violation of domestic and international ethical and moral/legal norms in regards to human experimentation—promoting and funding, approving human experiments that resulted in uninformed subjects being involved in experiments at 10 domestic and 6 foreign medical research institutions where they were intentionally observed while inhaling small particle contaminated air while being observed for adverse effects. These experiments carried out by prominent Medical Schools, is in spite of US EPA public pronouncements and testimony before congress that small particles are toxic, lethal (Hundreds of thousands of deaths annually) and carcinogenic.

US domestic law prohibits human experiments that might harm and international medical ethical standards for human experiments prohibit human experiments with no exceptions except exigencies of great need if the researchers act as subjects. Any other human experiments with a risk of harm are prohibited, and no consent will remove that proscription.

In the past 3 decades US EPA air quality research has been an abomination, relying on junk toxicology/epidemiology and the precautionary principle. The submitter has actively tried to expose the misconduct.

The proposal by the US EPA, discussed here to force EPA scientific transparency and scientific integrity is salutary and significant in all its elements, and the submitter is grateful for the change from the formerly fraudulent toxicology and epidemiology of the EPA to impose a new form of integrity.

I will detail in this submission the nature of the EPA sponsored research fraud, the methods and data manipulation and management that have resulted in EPA fraud on the public about air quality health effects, toxicological claims in other areas of EPA responsibility and the EPA full blown commitment to the hoax of CO2 levels as a cause of catastrophic warming. In these three areas of EPA research and policy making it is easy to identify the frauds on the public that are supported by a well-paid band of hired researchers and an in house gang of committed environmental true believers. The result is a fraud and research and policy conduct that is so badly informed and poorly researched and developed that it includes systematic commission of civil and even criminal acts to further an EPA agenda of aggressive environmental regulations that have created tremendous economic burdens for no good reason other than a fanatic environmental ideological agenda.

I will elaborate with specific references and documents in the submission below—elaborate on the irresponsible and flagrantly unscientific research funded and promoted by the USEPA on all matters of toxicology and epidemiology and my admonition to any reader is that if we do not stop this junk science for politics and ideology, we will follow the path of fools for a cause—the path of true believers that is paved with confirmation biases and fallacious science and policy making that violates the law and cheats the taxpayer in two ways, scaremongering, and regulatory burdens that steal resources and assets for regulatory compliance that diminishes better use of those resources in the public and private sectors. My promise to the reader is I will show you how and how much the EPA disrespects and abuses the rules and methods of science.

1. Other commentaries I agree with highlighted

The Washington Post
By Robert Hahn
May 10, 2018

https://www.washingtonpost.com/opinions/many-mocked-this-scott-pruitt-proposal-they-should-have-read-it-first/2018/05/10/31baba9a-53c2-11e8-abd8-265bd07a9859_story.html?noredirect=on&utm_term=.f7bcbc0a1887

Robert Hahn is a visiting professor at Oxford University's Smith School of Enterprise and the Environment and a non-resident senior fellow at the Brookings Institution. He recently served as a commissioner on the U.S. Commission on Evidence-Based Policymaking.

When Environmental Protection Agency Administrator Scott Pruitt proposed a rule last month to improve transparency in science used to make policy decisions, he was roundly criticized by interest groups and academics. Several researchers asserted that the policy would be used to undermine a litany of existing environmental protections. Former Obama administration EPA officials co-wrote a New York Times op-ed in which they said the proposal "would undermine the nation's scientific credibility." The Economist derided the policy as "swamp science."

But there is a lot to cheer about in the rule that opponents have missed. A careful reading suggests it could promote precisely the kind of evidence-based policy most scientists and the public should support.

Critics typically argue that the proposed regulation would suppress research that contains confidential medical records and therefore scientists could not share underlying data publicly for privacy reasons. Such restrictions, these critics say, would have excluded landmark research, such as Harvard University's "Six Cities" study, which suggested that reducing fine particles in the air would dramatically improve human health and helped lead to more stringent regulation of fine particles in the United States.

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But it appears that few defenders or opponents of the proposal have actually read the proposed EPA regulation, which is only seven pages long. Both sides distort the regulatory text.

Here's what the rule would actually do about the question of confidentiality of Personal Health Information under HIPAA or any other rule.

First, it would require the EPA to identify studies that are used in making regulatory decisions.

Second, it would encourage studies to be made publicly available "to the extent practicable."

Third, it would define "publicly available" by listing examples of information that could be used for validation, such as underlying data, models, computer code and protocols.

Fourth, the proposal recognizes not all data can be openly accessible in the public domain and that restricted access to some data may be necessary.

Fifth, it would direct the EPA to work with third parties, including universities and private firms, to make information available to the extent reasonable.

Sixth, it would encourage the use of efforts to de-identify data sets to create public-use data files that would simultaneously help protect privacy and promote transparency.

Seventh, the proposal outlines an exemption process when compliance is "impracticable." Finally, it would direct the EPA to clearly state and document assumptions made in regulatory analyses.

(It is this submitter's position that the privacy/confidentiality issues raised by EPA sponsored researchers are intended to be a distraction—there is nothing that prevents a release of the data used in the EPA studies of the 90s, and since, because data can be collected without identifiers that penetrate to reveal Personal Health Information or the identity of individuals. Most of the EPA Sponsored studies on small particle effects are death studies—there is nothing that is sacred and confidential about a death certificate. The Studies the on ozone effects can easily be done to redact personal health information.) Here's what the EPA's new proposed rule wouldn't do: nullify existing environmental regulations, disregard existing research, violate confidentiality protections, jeopardize privacy or undermine the peer-review process.

Taking steps to increase access to data, with strong privacy protections, is how society will continue to make scientific and economic progress and ensure that evidence in rule-making is sound. The EPA's proposed rule follows principles laid out in 2017 by the bipartisan Commission on Evidence-Based Policymaking — humility, transparency, privacy, capacity and rigor — and moves us toward providing greater access to scientific data while protecting individual privacy.

Instead of throwing stones, the scientific community should come together to offer practical suggestions to make the rule better. For example, the rule should recognize the incentives for scientists to produce new research. . . .

Done right, this could improve government policy not only in the United States but also around the world.
It's still hard to tell how this rule will affect EPA decisions, but one thing is clear: The rule will make the evidence by which we make policy decisions more transparent. The policy might not be perfect, but its benefits will likely far outweigh its costs.

Comment by the submitter—this is blowing smoke and making excuses for the "secret science" that has dominated EPA activities for decades—there is no privacy or confidentiality problem with gathering data on deaths in studies that do not depend on personal data or release that personal data. Death Certificates with accompanying information do not violate confidentiality or privacy rules and can be used to assess the validity of the studies submitted—period.

Michael Dourson is a prominent toxicologist in the private sector. Here, again below I provide bold highlighting for his essay on the matter of the transparency proposal:

From A Risk-Assessment Perspective, EPA Getting Rid Of 'Secret Science' Makes Sense

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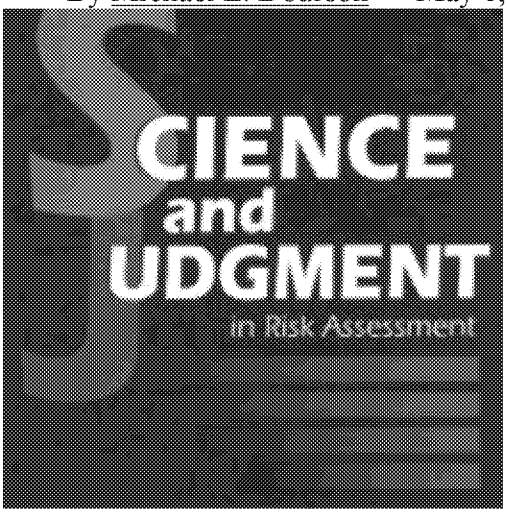
By Michael L. Dourson — May 8, 2018 Washington Examiner



Science and Judgment in Risk Assessment - the "Blue Book"

Environmental Protection Agency Administrator Scott Pruitt's recent announcement that EPA will not use "secret science" — that is science for which the underlying data is not available — is challenging. Whereas EPA is routinely in receipt of unpublished toxicity studies for chemicals designed for commerce, not all important scientific findings are publishable. Nor do scientific journals generally have sufficient space to include all data.

Much has been made in recent weeks of this new EPA policy, including an op-ed opposing it by former EPA Administrator Gina McCarthy and former acting Assistant Administrator Janet McCabe.

The media coverage has focused attention on how science is considered acceptable and useful in EPA's rulemaking. But missing from this is the perspective of risk scientists charged with protecting public health. In the case of EPA, it is often not enough for any one positive study to be published in a peer-reviewed journal. Such work often needs replication because a positive finding occurs, on average, in one out of every 20 studies due to chance.

If a study cannot be replicated, then it at least needs to make sense within the pattern of available data. For pesticides regulated by EPA, these data are often from hundreds of studies done according to federal guidelines.

Studies that are not replicated or that do not make sense in an overall pattern are still considered, however. Risk scientists will often contact the authors to obtain additional information in order to conduct their own analysis, a common practice within EPA.

When such data are forthcoming, without the need to break confidentiality or disclose confidential business information, independent analyses can be conducted and the public health is better served. But when such information is withheld by the authors, government risk scientists are often left with a dilemma.

For example, imagine that a series of studies come out on a single human group that is exposed to a commonly used insecticide, and they show an unexpected effect at extremely low exposures. This finding

has not been replicated and clashes with multiple animal and human studies that point to danger only at much higher exposures.

In this case, EPA scientists would ask the authors for the underlying data to confirm this unexpected low-dose effect. But let's say they can't get it. EPA is then left with neither confirmatory studies, nor information that makes sense in light of other studies, nor the ability to conduct its own analysis. Understandably, Pruitt has chosen a policy of not using such studies.

There is one sense in which McCarthy and McCabe are spot on. The judgment over which epidemiology and/or toxicology data to use for risk or safety assessment purposes should be left to risk scientists. But from my perspective as a risk scientist, Pruitt's decision is still correct. The public's interest is best served when science is replicable and consistent with other information. When studies cannot be replicated or are inconsistent with other information, access to their underlying data is vital to independent analysis. When the underlying data are not provided to a risk scientist, it is difficult to use this study to make a credible risk judgment, much less national rulemaking.

In short, the public is often worried about chemical exposure, as they should be when such exposure exceeds a safety level. But the public's interest is best served by trusting in experts dedicated to public health protection, not by withholding scientific data from independent analysis.

This article is republished from the *Washington Examiner*. Read the original here.

By Michael L. Dourson

Michael L. Dourson, PhD, DABT, FATS, FSRA, is prominent and accomplished toxicologist.

2. Choices in Risk Assessment--Report for the DOE 1994 by Steve Milloy

Steve Milloy, the founder and main writer for JunkScience.com for 20 plus years wrote a major research Monograph on Toxicological Policy issues as a contractor for the US Department of Energy in the early 90s. He exposed the federal agency science and policy misconduct in a major report "Choices in Risk Assessment" completed in 1994 on EPA 'science policy' and 'default assumptions.'

What is science policy? From "Choices in Risk Assessment", below are 10 common science policy issues and default assumptions used in EPA risk assessment.

Click here for a PDF of "Choices in Risk Assessment." It is more than 200 pages that explain why the EPA has lost its way, had lost its way in the early 90s because of ideologically energized environmental nonsense science.

Following the end of the Cold War, the Department of Energy (DOE) faced clean-up costs for its nuclear weapons sites amounting to hundreds of billions of dollars. The high costs would largely have been incurred because of EPA standards that essentially would have required the former weapons sites be returned to "Garden of Eden" status.

At the time, the DOE took the EPA standards so seriously that it was actually developing essentially a giant vacuum cleaner to suck-up the top layer of sand at the Nevada Test Site (approximately 5,400 square miles in size), decontaminate it and replace the sand.

Overwhelmed by the magnitude of the clean-ups, the Bush administration DOE commissioned Milloy in 1992 to lead an investigation into whether EPA clean-up standards were based on science or politics. Milloy's team of science and policy experts (called the Regulatory Information Analysis Project) compiled a report titled, "Choices in Risk Assessment: The Role of Science Policy in the Environmental Risk Management Process."

Completed in the fall of 1994, the report concluded that environmental policy was largely based on politics, not science. But when the report was completed and circulated for review within the Clinton administration-run DOE, the report was flagged as politically incorrect and Milloy was ordered by Clinton appointee Carol Henry (a former EPA staffer) to keep the report secret.

Sacrificing his business relationship with the Clinton DOE, Milloy disobeyed the order and released the report, which was subsequently featured in a Wall Street Journal editorial.

The attention that "Choices in Risk Assessment" garnered coincided with the Republican takeover of 104th Congress and congressional focus on regulatory reform, vaulting Milloy into the regulatory reform debate about to take place on Capitol Hill. Milloy testified before the U.S. Senate about risk assessment in the context of DOE clean-up on March 6, 1995. The DOE never wound up spending hundreds of billions of dollars to clean up its weapons sites. No word on what ever happened to the giant NTS vacuum cleaner.

Dunn comment:

The DOE report by Milloy

CHOICES IN RISK ASSESSMENT: THE ROLE OF SCIENCE POLICY IN THE

ENVIRONMENTAL RISK MANAGEMENT PROCESS

Prepared for Sandia National Laboratories

Sponsored by the U.S. Department of Energy

Office of Environmental Management and Office of Environment, Safety and Health (1994)

There are more than 200 pages, so I provide some pertinent sections emphasized by Milloy that pertain to scientific issues that hit on the big issues.

This is the link to the document:

https://junkscience.com/wp-content/uploads/2018/05/Choices-In-Risk-Asscssment-v-01-01Interior-With-Cover.pdf

Below is the table of contents, that will give one a sense of the magnitude of the report.

Hereunder I also feature sections of the summary and conclusions of the report—that are stunning. Consider—this is a report written by one man, essentially, a biostatistician and lawyer, and it exposes the problem of risk management in a politically charged atmosphere—the scaremonger environment of the US federal agencies committed to the environmental cause. Misconduct of the federal agencies and their paid researchers in matters of toxicology/epidemiology/risk management.

My question would be—why didn't this author get a National Award for exposing scientific malfeasance and scaremongering in Federal Agencies in the early 1990s? Well the answer is found in an analysis of what has gone on since—Deep State, totalitarian, junk science fraud that promotes the precautionary principle approach in all matters of public health research and science, the agenda of leftist environmentalist fanatics.

The proposal for scientific integrity and transparency is long overdue and the damage done by ideologues in the federal agencies will require a major overhaul in methods and internal review processes.

Much of the damage could have been avoided, had Milloy's report been respectfully considered and used as a guide for federal agency risk management. Instead it was suppressed and ignored by the Clinton Administration environmental fanatics.

CONCLUSIONS AND RECOMMENDATIONS

Conclusions

Many risks to human health and the environment are "unprovable."

Some risks to human health and the environment are provable. Provable risks can be measured or observed directly and include actuarial risks such as those associated with highway or air travel accidents. In contrast, other risks—such as those associated with low-doses of radiation or exposure

to chemicals in the environment—are often too small to be measured or observed directly with existing scientific methods and available resources. Additionally, specific health and environmental effects are often difficult to attribute to specific causes because other competing causes cannot be excluded with reasonable certainty. Such risks are unprovable. However, the fact that a risk is unprovable does not mean that it does not exist. Provable risks can be calculated, whereas unprovable risks can only be estimated through the risk assessment process. Although unprovable risks may be estimated and expressed in probabilistic terms, they are at best educated guesses and do not constitute knowledge or uncontroverted fact. In other words, the ability to produce a numerical estimate of an unprovable risk does not mean that the risk is proven.

Science policy issues are unavoidable in, and science policy decisions are essential to, the regulatory risk assessment process.

Risks are unprovable because of significant gaps and uncertainties in scientific knowledge, data, and method. When risk assessment is used to estimate unprovable risks, these gaps and uncertainties become science policy issues. Both risk assessors and risk managers make science policy decisions in order to bridge the gaps and uncertainties. Thus, science policy decisions enable the estimation of unprovable risks.

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CHOICES IN RISK ASSESSMENT

The existence and extent of science policy in risk assessment are rarely fully and fairly disclosed.

. . .

The lack of disclosure causes risk assessment results to be communicated essentially as fact. Such communication is misleading. Lack of full and fair disclosure of the role of science policy in risk assessment is not the fault of regulators alone. Media communication of risk information tends to omit discussions of science policy because such discussions: (1) do not fit into sound bites; (2) tend to detract from the sensationalism of the risk information; or (3) are not simple to communicate, and subtleties are lost.

Science policy decisions are responsible for regulatory programs and regulatory impacts that are justified on the basis of risk assessment

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CONCLUSIONS AND RECOMMENDATIONS

As in the risk assessment process, science policy and other assumptions play a significant role in the estimation of benefits and costs associated with regulatory programs.

When risks can only be estimated, the benefits of regulatory programs to reduce those risks also can only be estimated, are not verifiable, and depend on science policy-based assumptions. Similarly, cost assessments often depend on assumptions, are uncertain, and cannot constitute uncontroverted fact. An important distinction between estimates of costs and benefits is in the certainty of their existence. Because it is not possible to prove with certainty the existence of unprovable risks, the existence of benefits from regulatory

programs also cannot be proven. In contrast, while there is uncertainty involved in cost assessments, such uncertainty is associated with the magnitude of the estimated costs, not their existence.

Science policy decisions can be made so as to result in desired regulatory outcomes.

The case studies of fluoride in drinking water, asbestos in consumer products, unleaded gasoline, and used oil are examples of decisions where science policy-based assumptions help to justify desired regulatory outcomes.

- In the case of fluoride in drinking water, the weight-of-evidence science policy decision that fluoride was not carcinogenic in humans supported the continued fluoridation of water, a highly valued and desirable public health measure. This science policy decision also helped maintain the credibility of the Public Health Service, which has been promoting the use of fluoride since the 1940s.

- In the case of asbestos in consumer products, the science policy decision to consider only the estimated cancer risk from asbestos brake products and not to consider the potentially offsetting safety risk from the use of non-asbestos brake product substitutes helped justify EPA's decision to promulgate a ban on commercial uses of asbestos.

- In the case of unleaded gasoline, the science policy decision that mechanisms of carcinogenicity varied between rodents and humans provided the basis for concluding that unleaded gasoline is not carcinogenic to humans. This science policy decision helped maintain the credibility of EPA's program to remove lead from gasoline.

- In the case of used oil, the science policy decision that used oil is not a hazardous waste facilitates used oil recycling. Labeling of used oil as a hazardous waste would have resulted in a burdensome cradle-to-grave regulatory scheme for used oil that might have undermined recycling efforts and increased pollution from illegal or improper disposal of used oil.

CHOICES IN RISK ASSESSMENT

For the foreseeable future, science policy will remain the key to all regulatory programs that rely on quantitative risk assessment.

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Recommendations

Policy makers, risk managers, the media, and the public should be made aware of the role of science policy in risk assessment and subsequent risk management decisions.

Although risk assessors are likely to be aware of science policy issues and decisions, the same cannot be said for policy makers, risk managers, the media, and the public. Risk assessors often fail to emphasize the existence and extent of science policy in risk assessment. Where the role of science policy is not explicitly explained, risk estimates may be erroneously communicated to policy makers, risk managers, the media, and the public as uncontroverted fact. Because these groups are unaware of the role of science policy, they often fail to inquire about its impact on risk assessment. Either failure may result in regulatory decisions that are made on an uninformed basis to an uninformed, misled, or unnecessarily alarmed public. Risk assessors

should ensure that such miscommunication does not occur. Policy makers, risk managers, and the media should inquire about the existence and extent of science policy.

The federal government should institute a mandatory training and continuing education program on regulatory risk assessment and risk management for policy makers, risk managers, risk assessors, and their staffs.

Communication of risk assessment results should emphasize the role of science policy.

Because risk assessments for unprovable risks are educated guesses, risk assessment results should never intentionally or inadvertently be presented as fact. Full disclosure of the role of science policy should accompany risk estimates wherever presented, including Federal Register notices, executive summaries of regulatory documents, press releases, and other public and media communications. Disclosure is ineffective if it is inaccessible, comprehensive, explicit, and understandable. Disclosure should attempt to address the following questions:

- **Is the risk of concern provable, and can it be calculated? If the risk is unprovable, is it because the risk is too small to be detected with current scientific methods or because competing risk factors cannot be sufficiently distinguished?**

- **If the risk is unprovable, or provable but incalculable, what are the gaps and uncertainties in scientific knowledge and data that preclude the calculation of risk?**

- **What science policy decisions have been made to bridge these gaps and uncertainties? For unprovable risks, what science policy decisions have been made that concern the existence of the risk?**

- **Could alternative science policy decisions have been considered? What would the impacts have been on the risk assessment of these alternative decisions?**

- **What are the implications for regulation of the science policy decisions made as well as the alternatives? Do alternative science policy decisions reduce or eliminate the basis for regulation? Does consideration of substitution risks or lifecycle risks affect the basis for regulation?**

Answers to these questions will facilitate understanding of the likelihood that a risk exists and its potential magnitude. Improved understanding will enable: (1) policy makers and risk managers to decide on a more fully informed basis whether and what resources should be expended to address the risk; and (2) the public and media to debate the issue on a more fully informed basis.

Risk assessment guidelines may help provide a framework for the use of science policy in risk assessment, but only if such guidelines are flexible and complied with in good faith.

Risk assessment guidelines can provide a framework within which regulators can make science policy decisions. Such a framework would provide the regulated community and the public with the "rules" for science policy decisions in regulatory risk assessment. . . . With respect to potential judicial review, although it will be difficult for a court to rule on the scientific merits of an agency science policy judgment, a court can rule whether that judgment has been explained adequately. Ultimately, the merits of the judgment will be evaluated, and the agency's credibility will be weighed in the court of public opinion as well as by the scientific community.

Precedent has been established, and agencies should be encouraged to give meaningful consideration to alternatives to the default assumptions used in risk assessment

Only when policy makers, risk managers, the public, and the media fully understand the role of science policy decisions in risk assessment can the "real" issue in environmental and public health protection be debated. We must determine what society is willing to pay to reduce or avoid risks to human health and the environment which have been identified and estimated using science policy rather than science alone. These risks may or may not actually exist. If they do exist, they are likely to be relatively small or indistinguishable from other risks. If risks are too small or indistinguishable, it likely will not be possible to know whether regulation produced any benefit. The open debate of the value and priority of regulating these types of risks will enable, but not guarantee, policy and regulatory decisions to be made on a fully informed basis.

3. Commentary on proposed new, more stringent EPA ambient air standards for 2006.

Submitted for consideration in the comment period to end April 17, 2006.

April 13, 2006

John Dale Dunn MD JD

Brownwood, Texas violations of epidemiological and toxicology scientific rules are a scandal that cannot be ignored. The Dockery 1993, Pope 1995, and Samet 2000 studies (see endnotes) and other studies of health effects of air pollution relied on by the EPA, all showed that large studies with adequate power could not demonstrate relative risk of any significance. The studies all showed effects less than ten percent, rather than the statistically and scientifically required 200 to 300 percent effect. It is astounding the EPA has the gall to announce an air pollution crisis and propose more stringent air quality standards when none of the studies the EPA relies on show and proof of health effects.

The EPA is obligated to educate the public on the clear evidence that air pollution may have aesthetic and cultural import, but that there is no air pollution health "crisis." The EPA and its sponsored and supported health effects researchers are now just raising their voice in this debate instead of trying to use science. The EPA air pollution health effects science is an emperor with no clothes, as discussed below.

This commentary challenges the EPA to show one study that proves that one person has died due to air pollution in America in this past 20 years. People die for various reasons, suddenly and not so suddenly, as will be discussed below. That reality eludes the work of numbers crunchers who slave at desks over death certificate information like Pope and Dockery. One doesn't die from an exposure to air pollution, one dies from failed medical therapy, arrhythmias caused by long term coronary disease, stroke, pulmonary embolism, which are not caused by air pollution. The Asthma problem is an increasing problem not related to air pollution, since the rate of asthma is increasing with decreasing air pollution. The deaths from asthma will be discussed below and have nothing to do with air pollution, it is a socioeconomic phenomenon. It is time to retire the air pollution health effects studies of crude death tallies and it's time for the EPA to stand down from this repeated use of crisis talk and aggressive pursuit of pure air—a religious campaign disguised as science in the public interest.

As a last and compelling consideration, this author is familiar with death in America. As an emergency physician, much more familiar with what kills people than economists and public health officials who don't know which is the business end of a ventilator and live in the world of death certificates and mortality data. People die for many reasons and under many circumstances in America, but air pollution doesn't kill them, even the worst levels of outdoor air pollution one might imagine in America don't create a toxic level, which reveals the other major flaw in the EPA crisis rhetoric, junk science toxicology that completely disregards any effort to define toxin or toxicity. That subject will also be dealt with herein below.

The scientific epidemiological and toxicological criticisms of the EPA health effects studies and policy making are:

1. The Dockery 1993 and Pope 1995 studies did not show valid evidence of death effects, since they showed a death effects relative risk below 1.1, a negligible relative risk that is 10 percent of the minimal relative risk all epidemiologists consider necessary for proof of causation. A 200% or 300% change in death effect is the lower limit. Some epidemiologists require relative risk of 4 or a 400% effect when evaluating poorly controlled cohort studies.

2. This relative risk problem cannot be overcome by EPA and health effects researchers emphasizing the misleading use of the term statistical significance, which is not a proof test, but a statistical reliability test. One can be statistically confident and reliable but absolutely wrong.

3. The EPA and its health effects researchers have consistently and persistently ignored the lack of proof of health effects in these studies, and have made public announcements and allowed media reports to proclaim that thousands are dying in America due to air pollution when the studies do not show any proof of death effect at all. Lying for justice or an environmental ideal does not make the lie any less dishonest.

4. The health effects research used by the EPA has consistently ignored the basic rules for toxicology and the well-known phenomenon of threshold for toxicity. Only at the EPA does straight line toxicology have any status, mostly because it avoids serious science. Main stream toxicology science is still committed to the idea of threshold of effect and the old saying—the dose makes the toxin. The EPA scientists in house know the truth, but again politics and a commitment to a policy/environmental ideal results in lies.

5. Under no valid scientific analysis retro or prospectively, can the EPA use the methodologies or the results of the Pope, Dockery, McDonnell, or Lipfert (see endnotes) studies to justify one more burdensome air pollution regulation, but there is strong evidence for rescinding the last round of Air Quality Standards.

6. The EPA has a mandate to act only on the basis of acceptable scientific evidence of health effects, and is obligated to abandon the precautionary principle approach to regulatory policy, a pathetic substitute for legitimate science and clearly a principle founded in politics, not science.

7. The EPA could never convince a Federal Court, operating under Federal Rules of Evidence 702 and the court dicta for expert and scientific testimony that the EPA air pollution health effects science is valid proof of anything. The Pope, Dockery and Lipfert or Samet studies cannot be massaged or misrepresented enough to create any proof of air pollution health effects. The studies show trends within an insignificant range and "associations," that are not evidence of proof of health effects.

8. Precautionary principles that are used by the EPA as stand-alone policy justification are nothing more than a dressed up version of anxiety, cannot pass muster for admissible scientific evidence in a Federal Court and ignore the reality of risk/benefit analysis.

9. Based on the information reviewed in this critique, the EPA must revisit old rigs, forgo new, more onerous and expensive regulatory interventions, and the EPA must suspend its rulemaking in air pollution until it can find valid and reliable science on health effects.

Toxic air pollution existed in the past, and still may occasionally occur in some places on the planet as a local phenomenon, as particulate and other noxious air pollution in industrial areas, from various sources. Certainly air in big cities, Pittsburg, Los Angeles, Houston, New York was fouled in the past by air pollutants and even when not toxic, was smelly and visible, but trends in air pollution in the past 30 years as reported and confirmed by the EPA, have all been positive, attributable to changes in industrial processes, regulatory efforts and cleaner petroleum and coal consumption. Any study or discussion of air pollution is focused on a moving, improving problem. However the public thinks the air is worse than ever and there is an air pollution health effects crisis, and that is the fault of the EPA, its favorite researchers, and the mass media, who love to scare the public, since EPA budgets and environmental organization budgets depend on the anxiety of the public.

The death and illness rates during smog and air pollution catastrophe periods in the past were affected by less effective medical management and heavier cigarette smoking but also significantly higher air pollution than exists anywhere in the United States today, for many reasons. Deaths from acute respiratory failure in the past were more common and less preventable, but that is an independent factor related to medical advances and not due to air pollution itself. Airway diseases, the main effect of any air pollution, were less treatable before the 1970s. Pulmonary Medicine has changed dramatically for the better since 1970. Many airway diseases were more dangerous in the past and medical therapies frequently failed to control disease and death. Medical expertise in respiratory illness and cardiovascular disease is changed, but Pope and Dockery still yearn for the good old days of killer air because it scares the public. Their research ignores the trends of the last 20 years and below I will discuss a conscious deception in the second half of the Pope research from NCI data. In addition the EPA air pollution researchers continue to ignore the weakness of their findings, hoping to keep alive the "deadly air" panic talk alive.

People die for lots of reasons in America, but not due to air pollution. Air pollution health effects researchers know that, but act as though nothing has changed. The EPA should carefully reevaluate the number of deaths that researchers claim are due to air pollution in the last 20 years, but the EPA has a conflict of interest. No air pollution crisis might mean reduced EPA funding. No air pollution crisis might mean no funding for the researchers and their support organizations.

The air pollution health effects studies are based on weak epidemiologic relationships and trends carelessly described without definition as "associations," or "trends." Well ice cream consumption and drowning or boating accidents are associated by season, but ice cream eating doesn't cause water accidents. Associations are not proof, they are observations of phenomena--clusters of events that may or may not mean something. Epidemiologists know these things and should be careful when describing data associations and trends within insignificant ranges like less than relative risk of 2, so that the reader or reporter won't mislead the public or a politician. However, the definitions are not forthcoming from the scientists and researchers because saying that there is no crisis of air pollution means no publications for air pollution researchers, no

invitations to swell events, no funding, no chance to pursue a political agenda and change the world, making your mother proud.

The uncertainties of the air pollution health effects studies, the weak relative risks and the methodological problems of the most influential of the health effects studies are so noticeable and remarkable that during this comment period the EPA should reassess what has gone wrong in air pollution health effects research. The EPA should assess how these weak studies have affected EPA policy and rule making. The EPA doesn't have the right to panic the public and political leaders with deceptive junk science in the service of religious and fanatic environmentalism.

DISCUSSION OF THE STATISTICAL AND METHODOLOGIC PROBLEMS OF THE SAMET, POPE, AND DOCKERY HEALTH EFFECT DEATH STUDIES.

Author's comments are in bold. Studies referenced are underlined and the cite is in the endnotes by name and year. Sorry to disappoint those who want numbered endnotes—not a formal paper.

J. Samet (Samet 2000) published in the New England Journal of Medicine, a study modeled after the studies of Pope (1995) and Dockery (1993). He compiled and studied deaths in twenty American cities over a period of years, and compared them with air pollution monitor reports for those cities.

Samet in this 2000 paper asserts the following:

--"the relative rate of death from all causes was 0.51 percent increase for each increase in the PM 10 (10 micron size particulates) of 10 micrograms per cubic meter." This effect is not proof of anything, and Dr. Samet knows it. Less than a 1-% death effect is a nonsense result in a big cohort study.

--"the relative rate of death from cardiovascular and respiratory diseases rises 0.68 percent for each increase of 10 micrograms per cubic meter" Trends of less than 1% inside of a meaningless range of relative risk less than 1.05? A serious epidemiologist would snicker?

--"we also analyzed the effects of levels of carbon monoxide, sulfur dioxide, and nitrogen dioxide in a fashion similar to that of the analysis of pm 10 levels. After adjustment for pm 10 and ozone levels we found little evidence that these pollutants had a significant effect on the relative rate of death." Hold it, hold it, Samet says that he can't find an effect, even itsy bitsy effects from ozone precursor and carbon monoxide, something the other EPA favorite researchers say are killing thousands? Samet is not helping the EPA here. What about those dastardly pollutants? We scientists and particularly toxicologists are smiling to see Samet make a fool of himself and by adoption of this weak and deceptive epidemiology, the EPA doesn't look too good either. This is the kind of research the EPA has been using in air pollution regulatory policy now for years.

--"We did not find an effect of ozone levels on the overall rate of death from all causes or from cardiovascular and respiratory causes during the full year periods. Ozone levels were positively associated with mortality rates during the summer months when ozone levels were highest, although the 95 percent posterior interval extended into the range indicating no effect of ozone levels on mortality." Might this non-Johns Hopkins man who owns no jacket with arm patches translate for the benighted—Samet says even ozone doesn't have a death effect in his study. Score so far on this paper—rational skeptics for people in search of truth 3, EPA and Samet 0.

--"We found no evidence that key socioeconomic factors such as low socioeconomic status affect the association between PM10 and the risk of death in linear regression models." Some might be surprised to know that Samet works at a School of Public Health and all Public Health research for the last 20 years has shown clearly that there is a socioeconomic effect that produces premature deaths. Skeptics now 4 and running away, EPA and Samet still 0. Socioeconomic noise cancels out air pollution effects; that's the way the epidemiologists put it.

--"Our analysis also did not address the extent to which life is shortened in association with daily exposure to the various pollutants." Well golly Dr. Samet, everyone dies, how can you talk about death effects if you don't measure whether deaths are premature? Skeptics 5, Samet and EPA still 0.

Additional comments by this author:

1. The rate of death changes in Samet's studies are less than 1%, which is epidemiologically meaningless and shows no respect for the relative risk of 2 (100%) or more, that all cohort studies have to show in order to be able to assert effect. Little effects, even in studies with good confidence intervals and lots of power, are still empty studies, make work exercises. Samet's study was a nothing, yet it got published in the New England Journal, so one must wonder about political and environmentalist agendas up in Boston. I suppose they are neutral on the environment and always demand valid research in support of political agendas. I suppose.

2. The study fails to age/sex adjust for the important analysis—premature death. How did Samet get published? Samet is asserting proof of effect at less than one two hundredth of what is required in epidemiology. Then he says he didn't bother with measuring whether air pollution caused premature deaths. This research is about acute death affects? At non-toxic pollution levels? There is no plausible biologic science to support the idea that non-toxic air pollution kills people. Samet is beyond redemption. He's in scientific denial, or he works for the EPA agenda and he will be funded until he is old and gray.

3. Low relative risks, below 1.2, are the results in Samet's studies and all the other EPA health effects studies. One study goes above 1.2, the Dockery 1993 smaller study at 1.26, since recalculated by Enstrom in his article, Enstrom 2005 to 1.13. Such weak and minimal findings are unacceptable for publication, much less serious EPA policy making. The EPA and the studies misuse the term statistical significance, trends or association if they mean proof. There is no proof in any of these studies of an air pollution health effect. These studies prove nothing in the relative risk ranges of less than 1.3, particularly in cohort studies of death certificates that are subject to serious confounding.

4. The failure by Samet to find any effect, even these minimal effects, from other air pollutants like nitrous and sulfur oxides (ozone precursors), ozone, and carbon monoxide should give the EPA cause to wonder about any further attempts to impose new ambient air standards. The EPA has noticeably ignored Samet conclusions about these pollutants, why?

5. Samet's assertion that socio economics do not effect death rates is a an extraordinarily faulty conclusion for a public health researcher, since his study only looked at average area incomes for the twenty cities; and there is a vast body of public health research that shows that socioeconomics independently are a significant factor in life expectancy. (Wong 2002, Fitzpatrick 2001, Lantz 1998).

6. Socioeconomics is a factor and would nullify the signal from air pollution effect, and could even be a cofactor in another way by causing poor indoor air quality from substandard housing and a higher rate of

smoking along with a higher rate of underreported smoking. For example the poor have outdoor jobs where they can smoke more, and culturally they may be much heavier smokers with more inhaling, a potential confounder. Such confounding might explain the Ohio and West Virginia data from Pope 1995. That's why relative risk has to be set high, to avoid the effect of confounders not seen or understood.

The Samet article includes cautionary notes on the limitations of the study's methodology. His caveats are applicable to the all the previously mentioned Pope and Dockery, favorite EPA studies on air pollution health effects:

1. "For the pollutants measured on an hourly basis we calculated the 24-hour average." Toxicologists cringe at that one.

2. "If the pollutants were measured at multiple locations in a metropolitan area, we averaged the data." Remember the basic principles of toxicology, if you're downwind from an air pollutant you're safe, how can he say these things with a straight face. You have to know the patient and the toxin and the dose to know anything much about the science. Population studies are very crude at non-toxic levels of exposure.

3. "Since the Environmental Protection Agency requires levels of PM 10 to be measured only every six days, data for ozone and other pollutants were generally more available on more days." Good grief, this is a sham, a toxicology study with exposures every so often in sub toxic ranges.

4. "We analyzed the effect of the day on which the pollution data were obtained (the current day, the day before, or two days before) on the association with mortality rates. The overall effect did not vary with the lag interval selected. We report data for a one day lag between pollution variables and mortality." This is the place where Dr. Samet shows he doesn't know anything about death. You could be sick to death in a hospital and I can keep you alive indefinitely until the family gives up—where do those cases fit in Dr. Samet's arbitrary lag time of one day? What about people who die in a bed at a nursing home and haven't been outside in two years? These public health wonks and economists who hate dirty air do research as if a death certificate signed by the local GP is a piece of reliable data on the health effects of air pollution. They are in dreamland.

Then Samet says they found a temporal-causal relationship -- astounding! He didn't find a causal relationship, but he can find a temporal relationship. Did he dredge and dredge until he found something to point at? What's he talking about? Who's to know when the blips in the data are differences of less than 1%? That's not about cause of death, that's about political agendas and a polemic dressed up as science that causes public anxiety.

The good Doctor continues.

5. "Data on levels of PM 2.5 (small particulates) are not yet available nationally, since a monitoring network for particles in this size range is currently being implemented." This writer believes that Dr. Samet is working the agenda for the "annuity." Small particulates are an annuity for the EPA and air pollution researchers because, along with ozone, dust will never go away. Those air pollution demons assure EPA power into the distant future and more regs and anxiety. Dust is bad. Dust is always going to be there. It's the perfect air pollutant for the EPA.

Samet and others in the air pollution junk science club just use the PM 10-micron data that is measured every six days as a surrogate for PM 2.5. The supportive press and academic colleagues forgive such a lapse since they are working on the agreed upon agenda.

6. "Our analyses also did not address the extent to which life is shortened in association with daily exposure to the various pollutants."

Extraordinary. If the endpoint is a death effect, then the study must analyze premature death in mortal man and assess acute events as a measure of effect and endpoint for acute and/or chronic disease. To determine premature death effect, age and sex adjusted death rates are the accepted methodology, but Samet is just doing death rates and he gets published in the New England Journal of Medicine? Politics and the right agenda trump science and peer review?

7. "The finding that the association between PM 10 levels and the risk of death was strongest for cardiovascular and respiratory causes of death is consistent with the hypothesis that persons made frail by advanced heart and lung disease are more susceptible to the adverse effects of air pollution."

Again they didn't show that at all, they showed less than a 1% effect on death rates. I thought these people were dying of air pollution caused illness, not acute effects of air pollution, which at current levels couldn't kill a canary. What gives? What gives is that Dr. Samet is clueless because he's a numbers cruncher for the EPA in cahoots with his friends in the spic and span air society. I know why people die and it isn't from air in America, or even from Air America. Air pollution comes in many forms but we are obligated to live with toxicology science, not anxiety. Living organisms don't die for the thought of a smoggy day or from a bad smell. Dr. Samet and his cottage clack of air pollution hand wringers should go to a hospital and see how and why people die before they do these desk analyses of death certificates.

Despite these caveats the Samet research group asserts in the conclusion of their paper:

"Our analyses provide evidence that particulate air pollution continues to have an adverse effect on the public's health and strengthen the rationale for limiting levels of respirable particles in outdoor air." Samet says nothing about the significance of their research showing no death effect from ozone, carbon monoxide, sulfur and nitrous oxides. That would certainly disrupt current EPA policy, and he avoids an admission that the relative risks and death rate changes he found do not reach epidemiologic significance.

This study by Samet is sham epidemiology/science, junk science with lipstick, and the deception and "newspeak" harkens back to junk science in the service of the King or the current tyrant. Pope, Dockery and Samet are the officials/magicians/astrologers/conjurers in the EPA court, providing the EPA regent with needed "expertise" to justify the latest edict.

Briefly we will discuss below Dr. Samet's mentors, the EPA's favorite air pollution haters, Drs Dockery and Pope, who work together and change places on the authors lists of their papers.

The Six City and Pope Studies?

Dockery (1993) and Pope (1995) did studies that were the model for the Samet study discussed above. The studies did do better than Samet, in that they measured relative risk of premature death by studying death rate with age sex adjusting. Both Dockery and Pope were unable to show significant relative risk of health effect. The Pope and Dockery studies were used in the mid 1990s to justify EPA Director Browner's "emergency" new ambient air quality standards on ozone and other pollutants. The resulting cost was

estimated by the Center for Study of American Business at Washington University, St. Louis, at more than 100 billion. The Browner action was taken unilaterally, in spite of protests from many agencies within the government and without the approval or support of EPA internal experts. This action was taken without proof of a health effect, since Pope and Dockery never showed an acceptable relative risk. They were limited again to Samet's "associations" and trends within meaningless ranges below a relative risk of 1.3.

There is a greater relative risk of whole milk causing lung cancer than the relative risk that the EPA has shown for air pollution. One might say that's because of some confounder—well duuuh, that's why relative risk has to be above a threshold of 2 and some say 3, so confounders don't make the epidemiologist look confounded. Samet, Pope, Dockery don't care, they're on a roll and have the support of the environmentalist zealots, and the EPA (whoops, that's redundant). Call public relations, the research shows air pollution is killing thousands. It causes CANCER.

This paper points out that the EPA and the researchers are cheatin', and Dr. K. Popper, famous philosopher of science favorably cited by the Supreme Court in the Daubert decision, says that science must be more serious and reliable than politics. Popper asserts that science must be based on proofs that are reliable. Popper even talks about what the air pollution research by Pope, Dockery and Samet and the spic and span society is—Popper says some "science" is so bad it can't be falsified. How does one falsify something that means nothing? Associations at the edge of or in the midst of nothingness is what Pope's and the other health effects studies assert should be the basis for society wide regulatory regimes. Breathtaking—no pun intended.

The EPA says that air pollution kills thousands, because air pollution kills thousands. That is a tautalogy, a common tool for junk scientists. IT IS BECAUSE IT IS. I write here to tell the EPA that their anxious pursuit of clean air is more about politics and power and anger with modern industrial society that is already cleaning up the air, more about the religion of environmentalism. That's why the crisis, without the deaths or the science is a political or a polemic tool, not science. Not nice to fool with science that way, particularly when there is a Federal mandate that the EPA insist on scientific integrity for policy making. The EPA should not be in the business of ginning up false crises and scaring mothers that their kids are going to suffer from the air just so that the bureaucracy will thrive at the Federal and State level.

The EPA cannot claim to be unaware of the failure to prove health effects by the insignificant level of relative risk in the Pope and Dockery studies. These are the most basic of epidemiologic rules. And no subsequent studies have rehabilitated the failures of the Pope and Dockery studies. Samet, as described above, just repeated the same mistakes and came to up with the same lack of proof of health effect, unjustified conclusions and excessive and activist recommendations.

The barriers to a good study on health effects of air pollution for Dockery and Pope were the same as for Samet,

1. mobile populations,

2. unreliable, non-continuous and fixed monitor information,

3. no monitor information on some pollutants all the time (2.5 micron particles for example) or part of the time (10 micron and others),

4. an attempt to assess long term chronic health effects of air pollution by death studies, an acute phenomenon,

5. death certificates and raw death data used without autopsies,

6. inside air quality ignored for populations living indoors, particularly during old age, advanced medical illness, and terminal illness,

7. But most of all, no biological plausibility because the deaths are in the setting of non-toxic levels of air pollution (the inane straight line effect toxicology of the EPA cannot continue to get a pass—it is advocacy at the expense of science).

The EPA in assessing the air pollution effects studies must revive Bradford-Hill Criteria for toxicology.

The Bradford Hill (BH) criteria for toxicology are elementary, and establish biological plausibility for toxin effects. They require the toxicologist to establish plausibility, dose effect, reproducibility, time relationship, and a pattern of predictable and observable effects. Sounds like good science, but that's only part of it. Karl Popper was referenced above as the guru of the philosophy of science, and master or curator of scientific principles. The Popper legacy of science rules are referred to reverently in the Supreme Court opinion in the Daubert v. Merrill Dow Case [509 U.S. 579 (1993)] on admissibility of scientific testimony. Falsifiability is the key. To be true science one must submit to the test of being proven wrong. Pope and Dockery study results can't be falsified because they don't even allow a legitimate assertion of proof. They are tools in the game of politics, not in the game of toxicology. The EPA is required by common sense and federal statute to apply the BH criteria in air pollution studies, and all other toxicology work, but instead this wildly deceptive use of small changes within insignificant ranges of effect is souped-up to become the reason the EPA must act, now, immediately, to save lives. The EPA is saving itself, but the air pollution regulations are not saving any lives because the research would show the lives lost with valid epidemiology, and it doesn't.

The only reason that the EPA can create a crisis from the Pope or Dockery studies if it holds its nose and just projects to the whole population of the United States, then relative risk of less than 5% becomes thousands of deaths, even though it fails to show proof of one death caused by the toxicity of air pollution. Not one death.

If the biological plausibility of air pollution causing disease and death consistent with the BH criteria was established or could be established, then EPA and air pollution health effects researchers like Pope, Dockery and Samet could rest with their laurels. If air really were a killer or a toxin, we wouldn't see these weak cohort studies from the EPA with itsy-bitsy relative risks, and the argument would be over.

The EPA is not the national agency or institute for the arts, culture, pleasantness and good smells, it has a serious public health responsibility and a federal mandate to find toxins with legitimate science, promulgate appropriate solutions for the public benefit and then assess the effectiveness of what it has done. None of those steps are being taken in the air pollution policy making of the EPA.

The air pollution health effects studies in America will never be able to show the required relative risk of 2 or 3. What was the EPA role in such deception?

The idea that seems to control the EPA policy making on air pollution in the past 15 years is--ignore methodology and statistical problems, science be damned, move on to the grand program of air purification.

Find the ultimate terrible pollutant that will never go away, even with all our regulations. That is why small particulates are so promising for the EPA, enough so that these health effects studies talk about small particulates without measuring them, or measuring them in only one part of the study and not everywhere. The project of demonizing small particulates is reflected in the Samet study. He makes strong assertions with extraordinarily weak evidence, but he goes to the meetings, he knows what the EPA is concerned about. With EPA leading and frequently funding the crusade—science and truth casualties are acceptable. Small particulates are the worst crisis in the history of air pollution, they might cause CANCER.

I grew up and still live on a farm. I consider dust a reality that cannot be regulated away, just like ozone is part of the Smoky Mountains. There is a form of air pollution that is now being generated by the EPA in its ozone and small particulates crisis project—it is composed of dust, water, methane, and biological particulates.

Joseph Shumpeter said that the first casualty of a commitment to an ideal is the truth. The second casualty, this author asserts, is the unwary taxpayer and public that depends on responsible government. Solzhenitsyn said "The simple step of a courageous individual is not to take part in the lie. One word of truth outweighs the world." The EPA has become a slave to the lie of junk science in health effects research because the agency is devoted to its own importance and the importance of its religious and political agendas. EPA dredges up and makes icons of the precautionary principle, the small numbers/large projections lie, small trends within meaningless relative risks in populations studies, the refusal to recognize basic toxicology concepts. The EPA is a rogue agency in need of a stand down and close internal inspection with regards to bad policy making on the basis of bad science.

The Killer Smog

In The New England Journal of Medicine, Dr. C. Arden Pope, clean air activist, and one of the EPA's all time favorite air pollution health effects researchers, describes killer air in Belgium in 1930, Pennsylvania in 1948, and London in 1952 -- and uses those incidents as examples of why he thinks there is good reason to pay attention to a study in that issue of the Journal that claims to show a causal relationship between non-toxic air pollution and children's pulmonary functions. Again the study he is supportive of shows no epidemiological proof, just "associations," which are nothing more than statistical cluster puffs in population studies subject, as pointed out above, to bias and confounders. But the key is the study includes two important things for environmentalist zealots, children, and air pollution. Most importantly this study, like all the air pollution health effect studies, is working in insignificant causation ranges of effects so Pope and the EPA can talk about little bitsy trends and associations and urge that something be done before children die on playgrounds. They talk of these numbers exercises like they foretell an apocalypse. Gather the elderly and children and go seek shelter from the air, says Dr. Pope, an economist who got in the air pollution health effects business because he hated the air in Utah—imagine if he had lived in New Jersey. Dr. Pope advises---Stop breathing, if you must.

People do not go out into the streets of America, choke and die. The days of the people of London and Pittsburgh wearing dark clothes to mask the effect of soot and smoke are gone. The public health hanky battalion wants Americans to think air is killing their children and old folks, but in America ambient air pollution did not kill anyone, last week, last year, or in the last ten years. The panicky talk has to stop and the EPA must stop being the sponsor of the lie. The medical journals have to put their scientist hats back on and stop wringing their hands about nonsense environmental crises. The EPA is so busy these days frightening people about their rat studies and the imagined effects of so many things. Hardly enough time in

the day to pursue air pollution, except the EPA has lots of staff and lots of money and much energy and religious devotion to the cause.

EPA Policy and Regulation Activity

Fredrick Bastiat is known for his "law of unintended consequences," best exemplified as the analysis of the Paris shopkeeper's broken window. Bastiat made a common sense observation that when government or individuals choose to spend money or act, it produces desired and undesired effects, always making a ripple within the society and economy.

Let us propose to the EPA that if asthma deaths are predominately in young adult black males in America because of poor compliance (McFadden 1997), due to cost and availability of asthma treatment for disadvantaged adult black males or some other socio-economic or political problem, the EPA would be foolish to work on parsing senseless air quality regulations in preference to better asthma health care. The EPA would not be a party to such nonsense, would it, to relieve the anxiety of anxious environmentalists or satisfy the EPA staff's need for power and control?

There are no free regulatory actions. Every choice has multiple consequences, and government interventions have effects unforeseen. The EPA takes taxpayer dollars for every jot and tittle, every phone call, every new grand idea of every zealous bureaucrat. Every dollar spent for the EPA's ideal of pure air comes from somewhere and is taken from somewhere else.

The EPA is charged with responsible health effects research and policy making. The questions raised in the mid 1990s and now are the same:

1. If relative risk is a well known measure of cause and effect in epidemiology, why does the EPA allow relative risk below acceptable levels of proof to influence policy making?

2. Considering that EPA regulatory activity is tremendous burden to the economy, and the air regulations have a cost effect measured in billions per year taken from the taxpayer. If socioeconomic factors are an undeniable influence on quality of life and life expectancy, then can weak and unacceptable health effects epidemiology as described above, be excused for some abstract ideal of pure air?

3. Can studies that measure acute events in any way be considered studies of cumulative health effects? Are these death studies that Pope and the other air pollution researchers insist on basically flawed and deceptive. The answer is yes.

4. If some of the studies can't eliminate confounders, does the EPA have the authority to impose an onerous regulatory regime on the American society on the theory that cleaner air is a worthwhile, even if it doesn't have any effect on health?

Enstrom Particulate Air pollution Health Effects Study of 50,000 elderly Californians. 2005

Dr. James Enstrom, in the attached article found in appendix A, studied deaths in elderly Californians in 25 counties. He found that the relationship between fine particulates and mortality was very weak during the 1973-2002, particularly after 1982. He also reviewed the cohort studies on health effects of fine particulates and mortality by Pope, Dockery, McDonnell, and Lipfert, and found that their results were fairly similar to his, with the weakest health effects being present during the most recent years.

Enstrom finds:

1. The relative risks, age and sex adjusted and homogenized, are close to 1.00 in his and the other death studies (Pope, Dockery, McDonnell, Lipfert) he reviews in Table 10—there is no proof of health effect shown from particulate air pollution in his or the other studies.

2. Pope's year 2000 16 year follow up to the earlier (Pope 1995) study of the same cohort (Pope 2002) shows a declining cumulative risk from 1.07 to 1.04, first half to second That means to all but the innumerate that the relative risk in the second decade is well below 1.04. Hello Dr. Pope, Helloooo EPA.

3. Enstrom points out there are substantial geographic variation between the California populations of his study and Pope's Ohio, Kentucky and West Virginia data. The potential for confounders should be considered. I know something about that, and people in those states aren't the same as people in Enstrom's study. They might live different lives from their fellow citizens in Lala land. That's what homogenizing and sampling in epidemiology is all about. Without the data from those three states, Pope's studies would be more epidemiologically insignificant than they are, if that were possible. So much for avoiding cherry picking and the admonition to chip off the edges of the data to norm a cohort analysis.

The important points of the Enstrom study: .

1. Deaths and air pollution relative risks were assessed for 25 California counties, a cohort of 50,000 elderly Californians, and 39,000 dead before the end of the study in 2002. The relative risks were measured with proper confidence and homogeneity.

2. Relative risk found was extremely small and insignificant, 1.04 in the first part of the study (1973-1982), then relative risk of death from air pollution disappeared altogether in the second part of the study (1983-2002). Which will it be EPA, a crisis or salvation from killer air.

3. For the entire period the relative risk was 1.01 Pulleeez, 1-% risk? That's a relative risk of 1.01. I am closer than that to being rich and good looking, like Michael Jordan. The results would have to be 2.00 to be proof of any health effect, 1.00 is no effect.)

4. This Enstrom study, like all the other studies that the EPA uses to analyze health effects, and supposedly to study small particulate effects, is limited by the lack of PM 2.5 micron monitors before 1979 and only limited monitors after.

5. No increased death effects of any kind were shown in the counties with higher levels of air pollution, eliminating any dose response effect (a favorite rhetorical tool of the EPA researcher group), that, some of the higher pollution counties had lower relative risks. So is air pollution good for you if you live in California? In this range of relative risk absence of trend is meaningless but Dr. Enstrom does the

prescribed exercise, since the air pollution cabal likes to do trending and associations. The idea of a trend within an insignificance is interesting to consider, for fun, but not for science.)

6. Table 10 in Enstrom's paper shows a comprehensive review of comparable relative risks from large (Pope, Enstrom) and small (Dockery, McDonnell, Lipfert) studies, showing that only the Dockery study published in 1993 in a small cohort shows a relative risk above 1.1 at 1.15. All the other studies show relative risk similar to Enstrom, in the range of 1.07 or less.

7. In table 10 a number of the confidence intervals cross 1.0, the cumulative relative risk of the Pope study for the second half is lost in the failure to separate out the second half, indicating there is a reason to believe that in the second half of his study 1990-98, Pope had a relative risk approaching an insignificant 1.01. I worry, sort of, about Pope hiding this bad trend downward of an already weak relative risk. Could one suppose he has revealed this problem to his friends at the EPA?

Suresh Moolgavkar comments

It would not be practical here to cover all the writings of Suresh Moolgavkar on the epidemiologic and methodology problems he identifies in the EPA air pollution health effects research and policy making, and this writer does not understand some of the subtleties. Dr. M's brain and pen are too capable for an adequate treatment here, by a mere emergency physician. Dr. Moolgavkar's recent in depth review and critique of EPA particulate and air pollution research and policy making is in Appendix B.

Moolgavkar 2005 wrote a commentary on Enstrom's paper for Inhalation Toxicology discussed above (see second part of App. A). He asked the rhetorical question "Can contemporary epidemiological and statistical tools reliably detect miniscule risks, particularly with strong risk factors as potential confounders?" (Dr. Moolgavkar is too kind. He politely avoids exposing the junk science, the obvious, that miniscule risks in a cohort study like the results in the Pope, Dockery and other studies show no health effects at all and talking about trends in those ranges is silly.)

Moolgavkar objects to the methodology of proportional hazards modeling because "it is highly unlikely that proportionality of hazards would hold over the entire period of time covered by these studies." (The long term air pollution health affects studies). He asserts that it can be argued that "the SO 2 effect wipes out the PM signal in joint pollutant models." He does not even address the Samet study showing no SO 2 effect, so even that problem may ignore the more basic one that is so apparent—there is no detectable causal effect between air pollution and death. Dr. M is operating with the assumption that SO2 still is on the top of the list of bad pollutants. No doubt it is more toxic than others, but again, we must repeat the toxicology commandment—the dose makes the toxin. The air pollution health effect studies relied on by the EPA are ridiculously weak and are used as silly substitutes for a lack of laboratory proof that the current air conditions cause disease. The health effects research of Pope Dockery and Samet is just an exercise in the traditional deception of the "data dredge," the tool of crisismongers.

What is the point of quibbling about miniscule, below threshold of proof, differences in a cohort death study, some slavish devotion to arithmetic? I benefit, I suppose from not liking higher math, in this circumstances, that's why I focus on the medicine and the proper analysis of death studies and why people die.

Moolgavkar (2005 See App. B) wrote a lengthy review and criticism of EPA policy in Regulatory Toxicology and Pharmacology that exposes the epidemiologic and toxicological problems of the EPA air pollution health effects research discussed above.

Moolgavkar asserts: "evidence fell far short of supporting a causal association between particle mass concentration and human health." He goes on "the results of observational epidemiology studies can be seriously biased, particularly when estimated risks are small, as is the case with studies of air pollution. The Agency (EPA) has largely ignored these issues." "I conclude that a particle mass standard is not defensible on the basis of a causal association between ambient particle mass and adverse effects on human health."

Although Moolgavkar allows that the EPA may be bending the science in an attempt to pursue the precautionary principle on particulates, the precautionary principle under a mandate of good science in the public interest is not good policy. It is the default position for making concerns, feelings and aesthetics into the basis for regulatory actions that cost society billions for compliance. However no sandal-footed environmentalist gang of enviro-religious concerned citizens can allow the EPA to reject science.

The EPA is prohibited by federal mandate from ignoring science in the pursuit of the precautionary principle. The precautionary principle is anti-science and irrational by definition. Health effects not showed scientifically trumps feeling, concern and governmental overreach. The EPA is mandated by federal law to halt the overreach of the air pollution crisis crusade until it can resuscitate science in the public interest.

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Moolgavkar 2005 Moolgavkar S. A review and critique of the EPA's rationale for a fine particle standard. Reg Tox Pharm 2005; 42:123-44.

App. A

Abstract only with one table added.

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Fine Particulate Air Pollution and Total Mortality Among Elderly Californians, 1973–2002

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Fine particulate air pollution has been associated with increases in long-term mortality in selected cohort studies, and this association has been influential in the establishment of air quality regulations for fine particles (PM2.5).However, this epidemiologic evidence has been questioned because of methodological issues, conflicting findings, and lack of an accepted causal mechanism. To further evaluate this association, the long-term relation between fine particulate air pollution and total mortality was examined in a cohort of 49,975 elderly Californians, with a mean age of 65 yr as of 1973. These subjects, who resided in 25 California counties, were enrolled in 1959, recontacted in 1972, and followed from 1973 through 2002; 39,846 deaths were identified. Proportional hazards regression models were used to determine their relative risk of death (RR) and 95% confidence interval (CI) during 1973–2002 by county of residence. The models adjusted for age, sex, cigarette smoking, race, education, marital status, body mass index, occupational exposure, exercise, and a dietary factor. For the 35,789 subjects residing in 11 of these counties, county-wide exposure to fine particles was estimated from outdoor ambient concentrations measured during 1979–1983 and RRs were calculated as a function of these PM2.5 levels (mean of 23.4 μg/m 3). For the initial period, 1973–1982, a small positive risk was found: RR was 1.04 (1.01–1.07) for a 10-μg/m 3 increase in PM2.5.For the subsequent period, 1983–2002, this risk was no longer present: RR was 1.00 (0.98–1.02). For the entire follow-up period, RR was 1.01 (0.99–1.03). The RRs varied somewhat among major subgroups defined by sex, age, education level, smoking status, and health status. None of the subgroups that had significantly elevated RRs during 1973–1982 had significantly elevated RRs during 1983–2002. The RRs showed no substantial variation by county of residence during any of the three follow-up periods. Subjects in the two counties with the highest PM2.5 levels (mean of 36.1 μg/m 3) had no greater risk of death than those in the two counties with the lowest PM2.5 levels (mean of 13.1 μg/m 3). These epidemiologic results do not support a current relationship between fine particulate pollution and total mortality in elderly Californians, but they do not rule out a small effect, particularly before 1983.

Table ten is present as originally published in a pdf file of the article. Attached.

TABLE 10 Relative risk (RR) and 95% confidence interval (CI) for long- term all- cause mortality per 10- μ g/ m 3 increase in PM 2 . 5 for U. S. cohort studies based on PM 2 . 5

data, circa 1980

PM 2 . 5 Study characteristics

Study (author, year)

Data period/ Mean (range)/ (μ g/ m 3)/ Cohort geographic definition/ Follow- up period/

Mean entry age for period/ Number entered in cohort/ Deaths in follow-up period/ RR (95% CI)

Males

Dockery et al., 1993 1979– 1985 19 (11– 30) 6 U. S. cities 1975– 1989 _ 50 3671 a 830 a 1.15 (1.02– 1.30) b

Pope et al., 1995 1979– 1981 18 (9– 34) 50 U. S. SMSAs 1982– 1989 57 130,310 a _ 12,400 a 1.07 (1.03– 1.11) b

McDonnell et al., 2000 1973– 1977 32 (17– 45) 9 CA airsheds 1976– 1992 58 _ 1347 _ 375 1.09 (0.98– 1.21) b

Lipfert et al., 2000 1979– 1981 24 (6– 42) 42 U. S. counties 1975– 1981 51 26,067 _ 4600 c 0.95 (0.89– 1.01) c

1982– 1984 22 (8– 41) 1982– 1988 57 _ 21,467 _ 6100 c 0.94 (0.90– 0.98) c

1982– 1984 22 (8– 41) 1989– 1996 63 _ 15,367 _ 5765 c 0.89 (0.85– 0.95) c

Pope et al., 2002 1979– 1983 21 (10– 30) 61 U. S. SMSAs 1982– 1998 57 _ 159,000 a _ 36,000 a 1.05 (1.01– 1.10)

Enstrom, 2005 1979– 1983 24 (11– 42) 11 CA counties 1973– 1982 66 15,573 4701 1.03 (0.99– 1.07)

1979– 1983 24 (11– 42) 1983– 2002 74 10,872 8831 0.97 (0.95– 1.00)

Females

Dockery et al., 1993 1979– 1985 19 (11– 30) 6 U. S. cities 1975– 1989 _ 50 4440 a 599 a 1.12 (0.96– 1.30) b

Pope et al., 1995 1979– 1981 18 (9– 34) 50 U. S. SMSAs 1982– 1989 57 164,913 a _ 8365 a 1.06 (1.01– 1.12) b

McDonnell et al., 2000 1973– 1977 32 (17– 45) 9 CA airsheds 1976– 1992 58 _ 2422 _ 568 _ 1.00 (assumed)

Pope et al., 2002 1979– 1983 21 (10– 30) 61 U. S. SMSAs 1982– 1998 57 _ 200,000 a _ 24,000 a 1.02 (0.98– 1.06)

Enstrom, 2005 1979– 1983 24 (11– 42) 11 CA counties 1973– 1982 65 20,210 4094 1.05 (1.01– 1.10)

1979– 1983 24 (11– 42) 1983– 2002 73 16,116 10,815 1.02 (0.99– 1.04)

Both Sexes

Dockery et al., 1993 1979– 1985 19 (11– 30) 6 U. S. cities 1975– 1989 _ 50 8111 1430 1.13 (1.04– 1.23) b

Pope et al., 1995 1979– 1981 18 (9– 34) 50 U. S. SMSAs 1982– 1989 57 295,223 20,765 1.07 (1.04– 1.10) b

Pope et al., 2002 1979– 1983 21 (10– 30) 61 U. S. SMSAs 1982– 1998 57 _ 359,000 _ 60,000 1.04 (1.01– 1.08)

Enstrom, 2005 1979– 1983 24 (11– 42) 11 CA counties 1973– 1982 65 35,783 8795 1.04 (1.01– 1.07)

1979– 1983 24 (11– 42) 1983– 2002 73 26,988 19,646 1.00 (0.98– 1.02)

a Obtained from supplementary data (Krewski et al., 2000).b Recalculated from published data (US EPA, 2004).c Obtained from the author.

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Commentary

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Fine Particles and Mortality

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In an interesting paper in a recent issue (vol 17, issue 14)

of the journal, Enstrom examined the association between fine particulate matter (PM) pollution and mortality in a cohort of elderly Californians. The analyses used proportional hazards

regression and after adjusting for age, sex, cigarette smoking, and other potential confounders, Enstrom concluded, "These epidemiologic results do not support a current relationship between

fine particulate pollution and total mortality in elderly Californians, but they do not rule out a small effect, particularly

before 1983." Enstrom's analyses were based on a sub-cohort of individuals enrolled in the first Cancer Prevention

Study (CPS I) conducted by the American Cancer Society (ACS). Enstrom's conclusion is consistent with the conclusions of a cohort study among veterans conducted by Lipfert et al.

(2000), but is at odds with the results from analyses of the second ACS cohort (CPS II) by Pope and others (Pope et al., 1995, 2002; Krewski et al., 2000), which reported statistically

significant associations between fine particulate pollution and mortality.

Every epidemiological study has weaknesses and limitations and, undoubtedly, both proponents and skeptics of the 'fine par-ticles cause death' thesis will find much to criticize in the studies

that do not support their conclusions. These discrepant results raise an important question, however. Can contemporary epidemiological

and statistical tools reliably detect miniscule risks, particularly with strong risk factors as potential confounders? All the cohort studies referred to above use proportional haz-ards modeling for data analyses. But is proportional hazards really the appropriate tool for these analyses? First, it is highly unlikely that proportionality of hazards would hold over the en-tire period of time covered by these studies. Statistical tests for departures from proportionality of hazards have low power. En-strom states that, in his analyses, these tests failed to reject pro-portionality of hazards. However, his finding of a higher relative risk associated with fine particles over the period 1973–1982 is inconsistent with proportionality of hazards over the entire

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I have discussed the original CPS II study (Pope et al., 1995)

and reanalyses (Krewski et al., 2000; Pope et al., 2002) in de-tail

elsewhere (Moolgavkar, 2005). I note here, however, that

the reanalysis by Krewski et al. (2000) of the original (Pope

et al., 1995) study (which considered no pollutant other than

PM), showed quite clearly that the pollutant most strongly as-sociated

with mortality was not PM but SO2.In fact, when SO2

was considered along with PM in the model for all-cause mor-tality, the coefficient for sulfates was reduced to less than a third of its original value, that for fine particles was reduced to a sixth of its original value, and both became statistically insignif-icant. It is also of interest to note that consideration of spatial correlations attenuated the PM coefficients to a much greater extent than the coefficients for SO2.Given the much stronger and more robust association of SO2 with mortality in the CPS II reanalyses, I find it surprising that this study continues to be taken as providing strong support for the PM mortality asso-ciation. It can be plausibly argued on biological grounds that SO2 could not be causally associated with mortality. But that still does not explain why SO2 wipes out the PM signal in joint pollutant models. This awkward fact has simply been dismissed as being irrelevant. In a more recent study of the CPS II cohort that doubles the follow-up time and triples the number of deaths, Pope et al (2002) reported significant associations between fine particles and oxides of sulfur with all-cause, cardiovascular and lung cancer mortality. Surprisingly, despite the findings in the Krewski analyses that SO2 was the pollutant most strongly as-sociated with mortality, no joint pollutant analyses were carried

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App. B

Abstract only

A review and critique of the EPA's rationale for a fine particle standard

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Available online 24 March 2005

Abstract I review the rationale for the Environmental Protection Agency_s 1996 fine particle standard, which was based almost entirely on the epidemiological data with neither support from Toxicology nor understanding of mechanism. While many epidemiological papers available in 1996 reported associations between ambient particles and adverse effects on human health, many others did not and the evidence fell far short of supporting a causal association between particle mass concentration and human health.

The literature appearing after 1996 further complicates the picture. The large studies that have appeared after 1996, such as National Mortality Morbidity and Air Pollution Study, and the reanalyses of the American Cancer Society II study, report risks that are substantially smaller than the risks reported in the 1996 Criteria Document and Staff Paper. Moreover, concerns about confounding by weather, temporal trends and co-pollutants remain unresolved. Other issues having to do with model choice have resurfaced as a result of reanalyses of critical data to address a glitch in a widely used software package for time-series epidemiology studies of air pollution. Finally, contemporary examples show that the results of observational epidemiology studies can be seriously biased, par-ticularly when estimated risks are small, as is the case with studies of air pollution. The Agency has largely ignored these issues. I conclude that a particle mass standard is not defensible on the basis of a causal association between ambient particle mass and adverse effects on human health. Such a standard may be justifiable on the basis of the precautionary principle, however. The Agency could argue that the Science raises concerns about current levels of air pollution, and that reduction of ambient fine par-ticulate matter mass, if it could be achieved without an increase in the level of the ultrafines, could have positive effects on human health. If the Agency justifies a particulate matter mass standard on these grounds then the debate over the form and level of the standard will, for all practical purposes, belong strictly in the Policy arena.

Keywords: Air Pollution; Particulate matter; Criteria document; Staff paper

4. Dunn submission on Ozone October 8, 2007

Subject: Comments on Ozone Standards 2007

Submitted via the a-and-r-docket@EPA.gov 10-9-07

Docket ID No. EPA-HQ-OAR-2005-0172

Comments by John Dale Dunn, MD, JD, Civilian Emergency Medicine Faculty, Carl R. Darnall Army Medical Center, Fort Hood, Texas, Policy Advisor, Heartland Institute, Chicago, IL. Member, Board of Scientific and Policy Advisers, American Council on Science and Health, New York, NY.

Corrected and revised final draft submitted 0915 CDT 10-9-07.

1. The EPA ozone science does not justify continued aggressive ozone regulation and a new lower 8-hour standard.

2. The observational air pollution studies and the weak exercise/ozone inhalation studies cited by the EPA show weak associations and relative risk less than 1.5, as well as lab results best described as non adverse. The study evidence cited by the EPA would not be admissible in a Federal Court because it violates basic epidemiology and toxicology scientific rules.

3. The EPA's own Clean Air Scientific Advisory Committee advised in the past that ozone effects research did not show adverse effects and the ozone standard should be left as is.

4. There is no EPA research that shows any benefits from the air quality improvements of the past 20 years. Is it that the EPA doesn't want to report any improvement, for fear it will jeopardize agency funding? Is it evidence that the air pollution wars of the past 20 years were against a PHANTOM MENACE? Are the weak population studies on air pollution weak for a reason--there was no killer air in America?

DISCUSSION

The EPA cited health effects studies are weak on adverse ozone health effects and weak generally on air pollution adverse effects.

The Scientific studies discussed in the proposal document are reviewed below. Although the studies are cited by the EPA to justify the ozone standard, they are not what the EPA commentary says they are. They do not excuse the old standard, or justify the new proposed ozone standards because they are a combination of weak observational studies and no-effect intense exercise/high ozone studies.

Commentary on some of the prominent studies:

1 Dockery DW, Pope CA 3d, Xu X, et. al. An association between air pollution and mortality in six U.S. cities. N Engl J Med 1993;329:1753-9.

Weak observational study that mentions, but does not control confounders. The results are small effects with relative risks of an insignificant magnitude that is proof of nothing.

2 Pope CA, Thun MJ, Namboodiri MM, et.al. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med 1995;151:669-74.

Like the Dockery study above, one of the EPA's most important studies for justifying air pollution regulations. This study is another example of weak epidemiology with weak relative risks and no correction for confounders.

Even after the congress passed a law sponsored by Senator Shelby, requiring Pope and Dockery to produce their data sets, they still dodge and feint, and have not complied. Pope and Dockery are still in the inside clique of EPA favored and sponsored epidemiologists. They continue unhindered and well funded by the EPA and other governmental grant sources friendly to an aggressive regulatory agenda.

3 Hrostman DH Ozone concentration and pulmonary response relationships for 6.6 hour exposures with five hours of moderate exercise to 0.9, 0.10, and 0.12 PPM. American Review of Resp Dis Nov, 1990; 142: 1158-63.

Even heavy exercise with ozone inspired above current limits shows little ozone effect and no disease. The effect shown was mostly subjective respiratory mechanical effect. Ozone makes air heavy and increases its suspended/solute load.

4 Samet JM, Dominici F, Curriero FC, et.al. Fine particulate air pollution and mortality in 20 U.S. cities, 1987-1994. NEJM 2000; 343:1742-9.

Study of cities that claims to know how many days it takes for air pollution to kill someone, then proceeds to find no kill effect from all the air pollution factors, including ozone and ozone precursors, except small particulates, but then admits that the small particle monitor information is not available for the study and that big particles were used as a surrogate. Breathtaking, but published by Dr. Samet's friends in Boston. Incidentally the EPA on its air web site now has announced that large particles are no longer monitored or controlled because they do not cause adverse effects, but the old studies that concluded the dangers of small particles admit they used large particle monitor data as a surrogate for the small particles, since small particle monitors only became available in the late 1990s.

5 Wong JD, Shapiro MF, Boscardin WJ, et. al. Contribution of major diseases to disparities in mortality. N Engl J Med 2002;347:1585-92.

Discussion of confounders in death studies. Apparently has not been read by EPA sponsored and in-house epidemiologists, since the proposal documentation of the EPA makes little mention of he problem of the studies that are relied on—they make assertions without caveats like they were environmental gurus.

6 Fitzpatrick R. Ed. Social status and mortality. Ann Intern Med 2001 134;10:1001-2.

Lantz 1998 Lantz PM, Lepkowski JM et. al. Low income was an independent risk factor for premature death after controlling for health behaviors. JAMA 1998; 279:1703-8.

None of the studies used by the EPA for air pollution regulatory strategies control well for socio-economic status. Some of the studies do nothing more than mention that average income and education were used over large areas. Very similar to the casual use of wide-area, even regional monitors as measures of exposure to pollution.

7 McFadden ER jr., Warren EL. Observations on asthma mortality. Ann Intern Med 1997;127:142-7.

Shows that asthma mortality is in a select group of patients and caused by under-treatment and socioeconomic factors.

8 Mcconnell R, Berhane KT Gilliland F, "Asthma in exercising children exposed to ozone: a cohort study, Lancet 359 (2002) 386-91.

Selective reporting of this study ignored the protective effect of ozone, (yes, protective) in the whole cohort while making much of a minimal evidence of detrimental effects in one group--kids who were in three sports. McConnell is part of the Gauderman group that specializes in studying air in Southern California and always finds detrimental effects, even though many times the methodology and the evidence of risk are questionable and weak.

9 Gauderman WJ, Vora H, McConnell R, et al. Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. Lancet (on line) Jan 26, 2007. www.thelancet.com.

This study by the Southern California group had two major problems--1. Very small pulmonary function differences, less than 5%, which is insignificant, and no real negative trend, since the trend line only existed because of one outlier. There was also a high drop out rate. 2. The study measured differences in groups up to 1500 meters, dividing by 500 meters except for a group within 300 meters. Research shows that air quality from roadways is at background by 300 meters. The air quality on Southern California roadways was reported by H. Zhu in Atmospheric Environment 2002; 36: 4325-35 and in Environmental Science and Technology 2006; 40: 2531-36. Gauderman's group is well sponsored by a division of the California EPA. Imagine their funding stream if they reported no roadway effects?

Studies and analysis ignored by the EPA

The EPA also refuses to recognize the research and analysis that contradicts the EPA air regulation proposals.

Lipfert FW, Perry HM, Miller JP, et.al. The Washington University—EPRI veteran's cohort mortality study: preliminary results. Inhal. Toxicol. 2000, 12 S4:41-73. (Insignificant air pollution health effects.)

Enstrom J. Fine particulate air pollution and total mortality among elderly Californians, 1973-2002. Inhalation Toxicology 2005; 17:803-16. (Very large and long term study shows no air pollution death effect, in fact a counter intuitive protective effect of air pollution in many California cities. This study essentially nullifies the weak studies of Pope and Dockery as well as other death studies that are used by the EPA to push tighter NAAQS)

Moolgavkar S. Let. Fine particles and mortality. Inhalation Toxicology 2006;18:93-4. (Refutes the EPA air pollution project dogma. Discussion of EPA overreach and excessive regulatory zeal.)

Moolgavkar S. A review and critique of the EPA's rationale for a fine particle standard. Reg Tox Pharm 2005; 42:123-44. (Expose' of the EPA's failure to use good science to justify its agenda to make current ambient air pollution appear to be a serious health risk for Americans.)

Schwartz, J. No Way Back: Why Air Pollution Will Continue to Decline, (Washington: American Enterprise Institute, 2003). (Discussion of declining air pollution and improving air quality.)

The situation is so bad that the EPA and its sponsored epidemiologists and public health toxicologists control the literature and the journals. Journal editors now ignore toxicology and relative risk rule breaking. A recent poll by the National Institute for Statistical Science indicates that epidemiology journal editors no longer require data set production, p value calculation adjustments for multiple testing, and compliance with the rule on relative risk. The epidemiology journals have become political commentary on the hot environmental and social issues of the day—a mirror on the mental state of the academy.

Federal Rules of Evidence

The persistent failure of research on ozone and other air pollution observational studies to meet the requirement for relative risk of 2 and the p valued calculations without adjustment for multiple testing are examples of pseudo-science. The measure of scientific integrity, however, goes outside the academic and journal community. The Federal Courts have a stake in reliable evidence and the Federal Trial Court Judge makes the call on admissible scientific evidence.

The Federal Judicial Center's *Reference Manual on Scientific Evidence*, 2nd Edition (2000, West Group), also free on line at http://www.fjc.gov) Chapter on Epidemiology, written by highly esteemed experts, including Leon Gordis, the former Chair of epidemiology at Johns Hopkins School of Public Health deals with various matters of admissibility. The Manual states, at page 384:

> The threshold for concluding that an agent was more likely than not the
>
> cause of an individual's disease is a relative risk greater than 2.0. Recall that a
>
> relative risk of 1.0 means that the agent has no effect on the incidence of disease. When the relative risk reaches 2.0, that implies that the agent is responsible (with certain qualifications noted below) and implies a 50% likelihood that an exposed individual's disease was caused by the agent.
>
> A relative risk greater than 2.0 would permit an inference that an individual plaintiff's disease was more likely than not caused by the implicated agent. Thus, a relative risk of 2.0 would permit an inference that an individual plaintiff's disease was more likely than not caused by the implicated agent.

There are no major studies of ozone health effects relied on by the EPA that show a relative risk 2 or more. In fact there is not, at this time, a way to design a study on ozone that will show evidence of any relative risk, because there is no end point to measure. Ozone is a benign molecule, and doesn't cause death or disease. Exercise studies with excess exposure are a house of scientific cards for any EPA effort to build a toxicology argument against ozone.

The only reason the EPA can use these studies with relative risk of 1.5 or less, and not blush or apologize, is a political climate of panic about the environment and collusion in the academic and journal community collecting around the non-scientific social science concept of the precautionary principle. Discarding the relative risk rule is necessary to the survival of the precautionary principle, since the scientific evidence on ozone and most other pollutants cannot be shown to reach the relative risk of 2.

Expanding the effect of the EPA with "susceptibility."

The EPA also misuses the concept of sensitive or susceptible groups to make any exposure a concern for regulation. Susceptibility allows the EPA extraordinary latitude. There is always someone who is really, really sensitive—therefore the EPA plans to play the sensitive game and will make the society pay, eliminating any target toxin, regardless of the cost of the ablution. The rational regulatory regime does not adopt such a nonsensical approach, but the EPA embraces the concept as an excuse to overdo.

Reviewing the EPA United States air quality map, there are presently very few unsafe air quality areas, but that map will deceptively and dramatically change if the new ozone standard is implemented, along with the nonsense of the susceptibility. It will make no difference whether the standard is 0.06 ppm or .07 ppm, the non-compliance expansion guarantees that the EPA will exist into eternity.

The EPA is no longer in the business of protecting the public health and preserving the environment, the new range of ozone standards is an example of an EPA attempt to redefine what the environment should be and assure itself agency immortality. The EPA wants the world to be a scrubbed down bubble with no dust and no ozone for its own purposes, with no consideration of the rules of scientific integrity or even the mission of the agency to protect the environment and the public. Next the EPA will be regulating nitrogen, which is toxic if found at too high a percentage in the air. Really toxic.

The EPA is consciously and intentionally pushing the limits of scientific concepts of toxicity and epidemiology and cheating on the margins with the help of aggressive and flexible toxicology and epidemiology research. At this point a responsible Federal Judge, properly informed by the Federal Judicial Center Reference Manual on Scientific Evidence, chapters on toxicology and epidemiology, would throw out the "evidence" the EPA is using for this round of ozone standards.

The EPA refuses to study the health effects of the air quality improvements of the past 20 years. Why?

The EPA, like most government agencies or political advocacy groups, lives or dies by the old H.L. Mencken maxim about practical politics, that the public must be frightened, and anxious to be led to safety. False ozone fears and air pollution anxiety prop up the EPA. The EPA and its allies in the environmental movement feed the irrational and uninformed concern that the public has about a declining air quality, in the face of contrary evidence of improving air.

Why is there no research from the EPA that shows a public health benefit from the 20-year improvement in the quality of the air in the United States? Is the health benefit there and not shown or is it possible that the ambient air of 20 years ago, including the ozone levels, was not toxic? Generally even a blind toxicologist can prove a toxic effect by showing that the removal of a toxin caused a benefit. If there are air quality improvements that the EPA documents in its monitor information, then there should be a corresponding improvement in the health of the public.

Los Angeles and Houston air have improved—why no research to show the benefits? Is the EPA a one trick pony—they can only talk panic and crisis and bad air. Good air is not in the lexicon, only bad air and assertions of people dying from bad air? The proof of benefit would be the logical scientific inquiry to show the value of EPA activity and tighter air standards. Where are those studies of benefit?

If there is no real change in life expectancy or quality of life from air quality improvements, what will the EPA do, more importantly what should the country and the society do? Fire the EPA for lying or malpractice? The EPA and its allies in state and local government agencies, and in the non-governmental

environmental advocacy sector would be decimated by reports that there is no crisis in the environment, never was. They would also be, incidentally, unemployed and unemployable as pollution sheriffs.

Air Pollution Trends and Policy

Some places in America will be naturally dusty; some places will have natural background ozone levels that create haze. West Texas exemplifies the first, the Smoky Mountains the second. Trends in air pollution, control of ozone and ozone precursors in the past 30 years have all been positive, yet the EPA does not and will not report any benefit or improvement and continues to aggressively and energetically pursue every opportunity to increase its regulatory empire and authority. The EPA even sponsors and funds non-governmental entities like the American Lung Association and other rabid environmental groups that sue the EPA to push more environmental intrusions. That raises a question about conflicts and influence peddling, and contaminates the very important debate about EPA responsibilities to protect health and preserve the environment and maintain a high level of integrity in its science and research.

The blow back on the latest round of EPA overreach in ramming down the ozone standard is the protest of reasonable people confronting a new regulatory burden based on weak science. Ten years ago the EPA Clean Air Scientific Advisory Committee advised the EPA that ozone could not be shown to produce adverse health effects at the standard then, 0.12 PPM. Even then the CASAC, which is inclined to favor EPA policy proposals as a creature of the agency, was reluctant to support the ozone standard reduction from 0.120 ppm to a lower number. Chairman George Wolff said " although the panel member's opinions differed, none supported the lower end of EPA staff's recommendations, and a majority of members stated a position which included . . . the present standard."

EPA Clean Air Scientific Advisory Committee

The EPA Clean Air Scientific Advisory Committee (CASAC) in the late 1980s pointed out that ozone respiratory effects were not "adverse" health effects, and the CASAC in the 1990s refused to support using the Pope and Dockery studies to justify new NAAQS in 1997, but now the EPA is less scientific or objective in its analyses. The CASAC of today has become an advocacy committee committed to EPA agendas, even advocating more aggressive EPA activity. The CASAC of today has not and cannot be objective about ozone issues, and the current CASAC commentaries are not objective science but advocacy for aggressive environmentalism, now and forever.

There is no explanation for the CASAC conduct of the past few years other than political commitment to the environmental movement and the precautionary principle. In the past the CASAC and other agencies were the only chance that fanatic EPA officials would be brought under control, but now the CASAC has gone to the political side and cannot be trusted to show objectivity. Any argument for more regulation is supported. They represent the politicization of environmental science. CASAC commentary on small particulates last year was over the top.

Only 6 of 21 CASAC members supported the small particulate standards in 1996, the CASAC in 1996 advised in favor of the standard for ozone remaining at 0.120 ppm. Times have changed, the CASAC is now no restraint on junk science, and the CASAC of today is predictably in favor of any new and more stringent standard.

There are many in America who believe that the air quality is worse now than ever. That is because they get no reliable information from the EPA. The EPA is no longer a public agency that protects the public, but a

political propaganda mill, intent on panicking the public and working an environmentalist agenda. Informing the public of the improvements in air quality would reduce public anxiety and EPA and environmental group funding. Environmentalism would suffer a setback as a movement. The EPA is intentionally giving the public incorrect information about the current air quality, creating more anxiety, pollution warnings and claims about deaths.

This proposed new ozone standard is part of the deception, since the day the standard goes into place the American Lung Association, the EPA and the usual environmentalist organizations like Sierra Club will announce a new dirty air crisis. This latest round for ozone standard setting appears to be an effort by an EPA and its allies to reinvigorate their position as protecting the innocent public from killer air. They offer the naïve members of he public the proposal to create a pristine environment, more pristine than even Mother Nature could produce.

Consider, instead the reality as described by an environmental regulation expert:

> The United States has made tremendous progress in reducing air pollution during the last forty years. Air pollution has declined dramatically since the 1960s and 1970s, and virtually the entire nation now meets federal health standards for carbon monoxide (CO), sulfur dioxide (SO2), and nitrogen dioxide (NO2). Many areas of the country still exceed health standards for ground-level ozone ("smog") and airborne particulate matter (PM), but both of these pollutants continue to decline as well. Half of the nation's ozone-monitoring locations exceeded the federal one-hour ozone standard in the early 1980s, but only 13 percent exceeded the standard by the end of 2002. PM measurement methods have changed a number of times during the last forty years, but all trend data show PM levels dropping. Average levels of PM2.5—the form of PM now of greatest regulatory concern—have declined by a third during the last twenty years. (Joel Schwartz, 2003)

A good example of irrational panic mongering is in the September 9, 2004 issue of New England Journal of Medicine, in which C. Arden Pope, an economist cum environmentalist, describes as a companion piece to another children are victims of bad air article, describes killer air in Belgium in 1930, Pennsylvania in 1948, and London in 1952 and proposes those incidents as examples of why he thinks there is good reason to be worried. Pope is always worried, although he can't show me one person in his studies who really died from air pollution. They died as members of the cohort and he counted them as dead from air pollution after he looked at their death certificates. That's not a proper toxicologic analysis, that's an association. People don't die on epidemiologist's desks from associations.

In America ambient air pollution did not kill anyone, last week, last year, or in the last ten years. The crisis of bad air is long past, and the real health effects from air are non existent, but won't go away because the EPA is too big and too influential and too aggressive to go silent.

I agree with the Chairman of the Texas Commission on Environmental Quality, Buddy Garcia, who said in his letter of September 25, 2007 to EPA Administrator Johnson that ozone non-compliance will be the rule rather than the current exception, if the new standard is put in place. Mr. Garcia points out that 0.06 is well

known to the EPA as a background level in many environments—and that such a standard is irrational and cannot be complied with in places like the Gulf Coastal Plain.

Chairman Garcia also points out a little problem that the EPA ignores, that ozone precursors are mostly a product of mobile sources, not point/stationary sources, so the penalties and costs will be imposed on cities and communities for things they can't fix. Why is it that the EPA appears to care little about Mr. Garcia's concerns and his appeals for sensible science and policy making?

Summary

The research used to justify the proposed new ozone standard does not demonstrate results that meet the basic rule for proof of detrimental health effects. In fact the consistent findings of the EPA ozone research is insignificant ambient ozone pollution relative risk and laboratory evidence of fleeting effects if humans or animals are forced to breath high levels of ozone and exercise.

Research studies have shown that low relative risk results and pervasive confounders make it very unlikely that the proposed new ozone rules will have measurable beneficial or protective health effects. The EPA has failed to show the previous reduction in ozone levels has produced any benefits.

The EPA should abandon this precautionary-principle driven and junk science justified new standard, and retreat from continued aggressive tightening of ozone and other air quality standards.

Conclusion and recommendation.

There is no health effects science that justifies the current ozone standard of 0.08 ppm, so I urge the EPA to reset the ozone standard at the more reasonable 0.12 ppm, pending evaluation of the ozone control program for termination. Ozone should go the way of large particles, no longer on the list of EPA targets.

Imagine a government control program that has an end.

Economic and political effects of adoption of the recommendation.

I project that billions of taxpayer dollars and compliance costs could be returned to the citizens as soon as the EPA gives up chasing ozone, a benign component of the natural world.

I also project that a chastened and re-dedicated EPA might,fter the end of the ozone campaign, eschew future goose chases, and focus on serious, non-political, scientific inquiries in the public interest.

11-15-07

5. Dunn Presentation to the Health Risk Assessment Subcommittee of the and Executive Committee of the US EPA Board of Scientific Counselors 2007, 2008

John Dale Dunn MD JD

November 15, 2007 in person Bethesda, Maryland

Committee members and staff,

My name is John Dale Dunn. I am an inactive attorney. I teach emergency medicine at the Carl R. Darnall Army Medical Center, Fort Hood, Texas.

I asked for more time to present in early October, but I will do the best I can with the 3 minutes allotted. Dr. Stan Young from the National Institute for Statistical Science, and Dr. James Enstrom, epidemiologist from UCLA, will follow my presentation on the phone. We are not professionally or financially affiliated, but we share a concern about EPA scientific activity and integrity. In the future we will ask for more time to discuss our concerns with the BOSC.

H.L. Mencken made the prescient observation that the goal of practical politics is to create a hobgoblin, and make the public clamorous to be led to safety.

1. The Federal Judicial Center's Reference Manual on Scientific Evidence, published in 2000, and provided to the committee, was written by experts like Leon Gordis and Bernard Goldstein.

2. The scientific advice and rules provided to judges in the Manual are generally held and well known to the committee.

3. My concern is that EPA research repeatedly violates the Reference Manual rules on observational study relative risk as proof of causation and the rules on toxicology. I think well established and reliable scientific rules should govern EPA research.

4. The Manual insists on Relative Risk of at least 2 for proof of causation in observational studies. The EPA sponsored and funded research repeatedly and flagrantly violates that rule and claims small effects are reliable.

5. The Manual recites the traditional rules of toxicology, including the concept of threshold. The EPA violates those rules by arguing for high-dose toxin experiments on hybrid homogeneous rats and mice, combined with linear modeling as proof of toxicity.

EPA Administrator Browner had the chutzpah to claim that the adoption of ambient air standards proposed in 1995, that were based on Pope and Dockery small effects results, would prevent 20,000 deaths.

The quality of air and the environment is better now, but Americans think the environment is worse due to EPA public relations and research activities.

The BOSC is charged with assuring reliable and credible EPA research and policy making. EPA science should not risk a sensible judge applying the rules and finding EPA research inadmissible. It should be research and that does not panic the public with weak and incredible claims, like those made by Ms. Browner.

The BOSC should prevent the EPA shouting "consensus," intimidating the academic and journal community into breaking the rules and creating unjustified public anxiety.

Respectfully,

John Dale Dunn MD JD

Previous submissions:

Dunn Comments on small particle standards--2006.

Dunn Comments on ozone standards--2007.

Reference Manual on Scientific Evidence 2nd Ed. (2000)

Submission with this email:

2001 Editorial by Drs. Samet and Burke in American Journal of Public Health defending use of small effects studies.

Amicus brief submitted on behalf of Drs. Wogan, Eaton and 29 other distinguished Scientists criticizing EPA Linear Modeling on dioxin.

DUNN PUBLIC COMMENT SUBMISSION

MEETING OF THE EXECUTIVE COMMITTEE

Board of Scientific Counselors (BOSC) OF THE EPA

JANUARY 24-25, 2008 by phone with written submission emailed.

In the recent months I have provided materials and commentary on scientific integrity issues that fall within the BOSC Mission. The submissions and commentary were to the HHRA meeting and NERL meetings.

I renew for the Executive committee, my concerns about the following:

1. EPA sponsored scientists have repeatedly used relative risk in the negligible range as proof of health effects causation, in spite of epidemiology rules to the contrary, as recited in the Reference Manual on Scientific Evidence, published by the Federal Judicial Center.

2. The same is true of EPA sponsored science on the issue of hi dose rodent toxicology combined with linear modelling with no threshold. Again, I submitted the Reference Manual chapter on toxicology.

3. In addition to the Reference Manual materials, I submitted the brief filed on behalf of the American Council on Science and Health and many distinguished scientists criticizing EPA linear modelling and no threshold toxicology.

I will not resubmit these materials today, since they are already available to the Executive Committee, in addition to submissions by Dr. Stan Young on multiple testing unreliability and Dr. James Enstrom's submissions on his concerns about conduct in the scientific community that stifles inquiry and penalizes legitimate scientists.

The Executive Committee is composed of members much more expert than in the problems of data dredging in small effects science. The EPA is also embarked on a new series of toxicology projects that will increase the chance for problems, the genomic effects toxicology and small effects chemical toxicology research projects that increases the risk of more uncertain and unreliable research in health effects.

I ask the Executive Committee to begin to make more inquiries in these areas, and hold the EPA to a higher standard of reliability. The BOSC represents the interests of the public in assuring EPA science does not just promote interests and agendas of the EPA, but a balanced and reliable effort on behalf of the public interest and deserving of the public's trust.

Thank you for your consideration.

6. Essay by John Dale Dunn for congressional Aides of the Space, Science and Technology Committee of the House, on matter of Science and the Law

10-10-11

Introduction to fallacious and erroneous science and the law.

In addition to reviewing the Reference Manual on Scientific Evidence of the Federal Judicial Center, txt and links in this folder, there are also some excerpts from a book by Peter Huber, PhD and attorney, and Ken Foster PhD on the meaning of the new rules of admissibility for scientific evidence and testimony.

The section of the book excerpted focuses on fallacies in science and the intellectual, epistemological, political, social and psychological aspects of bad science.

First, however, anyone attempting to understand the current state of affairs should read the folder file on Angelo Codevilla, the essay on scientific pretense, along with the farewell speech by Dwight D Eisenhower in 1960 that discussed after the military-industrial complex, the government-research complex and in that section Ike warns of the danger of big government funding research programs and how such developments might corrupt the scientific process, which is not about authority and consensus, but skepticism and humility, the self-questioning that is essential for good science.

After reviewing the essay by Codevilla, one might expand on the problem of oligarchies in the other essay by Angelo Codevilla on the Ruling Class in America, that discusses the problem of elitist oligarchy dominated government tainted by group think and statist agendas. That is critical to the development of science in the service of politics.

Peter Huber, Kenneth Foster *Judging Science* (1997 MIT Press)

The chapters of importance in this book discuss the judicial articulation of what is good science, then essays and discussion on '

Testability and Falsification—Chapter 3

Errors in Science—Chapter 4

Reliability—Chapter 5

Scientific Validity—Chapter 6

That's enough for this folder material and will be summarized with the excerpts from the book including in the materials of the folder.

The materials are valuable, because they include original essays by many of the important figures in the philosophy of science. This summary is by John Dunn, but the original writers are better in their original discussion for more in depth inquiry.

1. Karl Popper is quoted and his teaching on good science is adhered to in the Blackmun Daubert opinion. Popper, a philosopher, emphasizes the importance of the deductive method of development of scientific concept and solutions, which is heavily focused on evidence and testing theories developed for evidence that might falsify the theory. Falsifiable is essential to a good scientific theory, otherwise Popper considers the theory non science. Pp. 35- 55

2. Weinberg proposes a concept of trans-science that is not practically verifiable or it may exceed the sensitivity of the instruments and methodology. Pp 55, 56.

3. An example of trans-science is epidemiology in the range below proof of effect, for example uncertain methodology or Relative Risk of less than 2. P 57.

4. Another concept of trans-science that is rhetorically in widespread use is to prove no risk, to prove the negative. P 58.

5. Reliability and validity are not the same, for example a reproducible and reliable measure may be invalidated because of a poor instrument or methods or bad underlying science. The first error is easier to identify and correct than the second, which looks valid. P 69-71.

6. Confounders produce validity errors and are the reason observational studies require effects of 100 percent—there are many confounders, listed at p 71, migrations or maturation of the study group, attrition, selection, regression to the mean, sequence of effects, experimenter and subject biases and behavior, even simple things like recall bias and overreliance on recall.

7. Confidence interval is another form of measure of reliability of the data, providing a range of accuracy or reliability around a result. P 79, 81. But some say that confidence interval is too loose. One important consideration is that if a confidence interval includes 1.0, there is no basis to argue for an effect. STUDIES RELIED ON BY US EPA THAT INCLUDE 1.0 IN THE CONFIDENCE INTERVAL (CI) ARE NOT RELAIBLE TO SUPPORT AN ASSERTION OF TOXICITY. A CONFIDENCE INTERVAL THAT INCLUDES 1.0 SHOWS A NULL EFFECT.

8. When the signal (results) is in the range of the noise (background natural variability) the reliability of the research is compromised by the signal to noise confusion. In studies with small effects like the US EPA air pollution premature death studies, confirmation bias (also called tunnel vision) energized by intellectual passion and commitment to a political agenda produce studies that do not justify the policies proposed and pursued or the regulatory regimes imposed. P 84.

9. Fallacies and fallacious thinking and research derive from reliance on authority, consensus, acceptance of a vote of those present, obfuscation or cover and selection bias in the service of intellectual passion or ambition, or the "gold effect" which is another form of intellectual passion combined with social

pressure consensus bias. All these biases and prejudices and fallacies of thinking are in contravention to the gold standard for scientific inquiry—skeptical experimentation by researchers who are the most strict judge of the nature and reliability of their research and disciplined in analyzing whether their evidence is proof of a theory. P 85.

10. Intellectual passion and ego of the researcher are sources of bad science and one of the most important conflicts of interest. Ego produces a failure to test one's theory adequately and produces confirmation bias—gathering supportive evidence and rejecting dissent or disagreement and evidence that falsifies the theory in favor. All researchers tend to mythologize themselves and their research, and lack the humility to recognize their own fallibility or see the limits or weakness of their research. Their investment in their career and stature make them rigid and uncritical in their assertions of theory or positing of solutions or answers. P 86

11. Sick science is characterized by:

a. The maximum effect is produced by a phenomenon of barely detectable intensity.

b. Observations are made near the threshold of visibility of the eyes or instruments.

c. There are claims of great accuracy (and significance).

d. Ad hoc excuses are used to nullify any dissent or criticism.

e. The supporters rise and then fall.

12. Another characteristic of sick science is the cargo cult syndrome—pretense of scientific methodology that has no substance. P 89.

13. Another characteristic of sick science is the reports of effects that are considered ominous are in the range of background. E.g. EMG that was proposed to cause terrible carcinogenic effects in the range of the earth's magnetic fields.

14. The pattern of error that goes to policy making, for example ignoring opportunity benefits, fear of introducing new technologies on the precautionary principle, ignoring safety risks associated with a proposed regulatory regime or remedy, ignoring large existing benefits in favor of fear of risk or the precautionary principle, or MOST IMPORTANT, IGNORING THE UNINTENDED CONSEQUENCES OF PROPOSED SOLUTIONS, EITHER IN TERMS OF COMPLIANCE COSTS OR DIRECT AND KNOWN RISKS AND DETRIMENTS.

15. Procrustean data torturing is not different from opportunistic data torturing, and certainly no less pernicious and deceitful. P 99.

16. The seven deadly sins of knowledge or the cognitive illusions that are nefarious;

a. overconfidence

b. magical thinking

c. predictability in hindsight

d. anchoring or tunnel vision

e. ease of deception

f. probability blindness or chance ignorance

g. the game of conjuring of linkages and ignoring the weak links in a chain P 118, 119

17. Reliability refers to the reproducibility of the data. Reliability is measured in terms of sensitivity and specificity. Bayes' theorem measures positive and negative predictive values that are both dependent on sensitivity and specificity. P 113-115.

18. Back to Popper, the soundness of a theory depends on

a. the conclusions must be internally consistent

b. avoid tautological statements that prove nothing but just reference the assertion

c. look for scientific advances in a theory

d. test a theory with experiments

19. The theory must be logically consistent, falsifiable, must assert something new, or novel, and it must be verified by experimental evidence (p 138, 139).

20. There are a fistful of fallacies

a. indirect cause asserted

b. necessary causes are not always sufficient cause

c. temporal or post hoc causation is not real causation

d. ecological fallacy transfers observations about populations to individuals

e. the faggot fallacy piles small and suspect items of proof or evidence and attempts to validate by the bundle or the height of the pile

f. weight of evidence fallacy is similar to e. and relies on the pile

g. bellman's fallacy is another form of the pile fallacy

h. fallacy of risk is the confusion of absolute and relative risk and using one or the other to deceive

i. inappropriate extrapolation is the assumption that one knows the trends and can project

j. new syndrome fallacy is novelty to an extreme

k. insignificant significance—overemphasizing the importance of statically significance in proof of a theory

l. Fallacy of ignoring large effects in small studies because hey fail a statistical significance test.

m. Positive results are fallaciously given more significance

n. Denial of medical mistakes (all these are on P 143)

21. There are good rules for reading and evaluating a paper as a reviewer. P 149-150

22. Feinstein dissects fallacious and alarming medical reports on reserpine causing breast cancer, coffee causing pancreatic cancer, and alcohol and breast cancer. Feinstein reviews how the studies on these reports were flawed. P 156.

It is important to note that the book Judging Science is an exceptional effort by extraordinary authors and this writer cannot do them justice. The books sections are excerpted by necessity.

Buying the book will be the best choice for anyone compelled to learn the intricacies of legal management of scientific evidence and the theories of science that underlie any reasonable discussion of scientific reliability and veracity.

7. An abbreviated story of the effort by John D. Dunn MD JD to expose the misconduct of the US EPA in matters of toxicology and epidemiology.

The Environmental Protection Agency's Particulate Matter Rules: One Physician's Crusade against Cargo Cult Science (JPANDS Spring 2014)

John Dale Dunn, M.D., J.D.

http://www.jpands.org/vol19no1/dunn.pdf

The U.S. Environmental Protection Agency has an annual budget of almost $10 billion, and influence and power far beyond that, with U.S. industry and society always subject to EPA orders, regulations, guidelines, fines, and edicts on environmental compliance.

My effort to expose EPA's bad science and policy making began in the early 1990s, and has culminated in the past 2 years in EPA's admissions, in declarations under penalty of perjury, that inadequate and unreliable, even unethical science underlies EPA regulatory regimes under the Clean Air Act (CAA).

In the infamous Tuskegee syphilis experiment, innocent black Americans suffered the depredations of advanced syphilis as federal public health agents denied them treatment. Now EPA-sponsored studies deliberately expose human subjects to pollutants that the EPA claims to be toxic, lethal, and carcinogenic. The Tuskegee experiment was unnecessary—the effects of advanced syphilis had been known for centuries. The EPA claims it already knows how dangerous fine-particulate air pollution is, but the agency is funding human exposure experiments with what EPA-published air quality standards say are toxic levels of fine-particulate air pollution.

Environmental Law Course

I was a small-town emergency physician and inactive attorney when the dean of sciences at the local Howard Payne University asked me to teach environmental law for the new undergraduate major in environmental science. I obtained the federal and state statute books and put the course on the curriculum to

include adult education for community people interested in compliance issues, as well as the environmental science students.

My study of the economics and politics of environmental regulation led to the conclusion that it involved a **form of cargo cult science** (fake science that looks like science), as described by Nobel Prize winner Richard Feynman,1 that develops when government money is lavishly given to people in the academy to support a political agenda built on a false threat of public harm. EPA's cargo cult science was in the area of epidemiology (population studies) and toxicology (study of poisons and harmful substances). It allowed EPA to beat the panic drum and scare people about killer environmental poisons that were not harming anyone in the ambient environment. This coincided with the growth of the radical environmentalist movement, which I would describe as a cult built on pantheism and a commitment to statist control of society.

One of my guest lecturers, an engineer responsible for compliance for Phillips 66 and an alumnus of Howard Payne, said that EPA would eventually take as much as five percent out of the gross domestic product. His predictions didn't seem so exaggerated when, in the mid-1990s, ozone air standards proposed by EPA Administrator Carol Browner under President Clinton were estimated by economists to cost the economy more than $100 billion. Browner pushed ahead in spite of objections and opposition by EPA's in-house Clean Air Scientific Advisory Committee, and all the Democrat administration-controlled executive agency divisions and offices.

Many aspects of junk science in the public health sector promoted by agencies like EPA are explained by biostatistician and lawyer Steve Milloy in his books *Science Without Sense* (Cato, 1995), *Silencing Science* (with Michael Gough, Cato, 1998), and *Junk Science Judo* (Cato, 2001). Other valuable books on bad science are by Peter Huber: *Galileo's Revenge* (Basic Books, 1991); *Phantom Risk: Scientific Inference and the Law* (MIT Press, 1993); and the most extraordinary study of junk science I have read, *Judging Science: Scientific Knowledge and the Federal Courts* (with Kenneth Foster, MIT Press 1997). The last focuses on the question of science as evidence and how rules of evidence should be used to determine admissibility of scientific testimony and evidence in court proceedings.

"Clean" Air vs. Safe Air: Justifying Regulatory Overreach

The cottage industry of air pollution research is committed to the proposition that air pollution panic is justifiable if it allows regulatory reach by the EPA that would satisfy an aesthetic demand for "clean" air. In my opinion, the research community is distorting the intent of the Clean Air Act (CAA), which should have been named the Safe Air Act since it is impossible to make the air"clean" of pollutants (such as dust, for example). The statutory language of the CAA required the EPA to identify harmful air pollution and mitigate the effects, not make the air "clean."

One of the most prominent EPA-sponsored researchers in air pollution is Jonathan Samet, M.D., chair of epidemiology at Johns Hopkins Bloomberg School of Public Health and chair of the EPA Clean Air Scientific Advisory Committee (CASAC). In a 2000 paper in New England Journal of Medicine, 2 he claimed that fine particles were causing deaths. This claim was based on an inadequately small association of fine particulates and deaths in a study of 20 cities. Small associations are not proof of causation and could easily be a random effect or result from data mining and dredging. By the year 2000 EPA had used its junk science to stack up a well-funded and sponsored pile of papers using the same bad methodology and claims

as the Samet paper, going all the way back to the Pope 3 and Dockery 4 foundational air pollution studies that created the EPA air pollution research and regulation crusade of the 1990s.

Samet and his fellow air pollution researchers, who had become advocates, would mine the data to find a small association and then announce a threat and crisis. In his 2000 paper, 2 however, Samet made an admission that I thought very important: he could not find a toxic effect from the other EPA criteria air pollutants, carbon monoxide, sulfur oxides, ozone, or ozone precursors such as nitrogen oxides and volatile organics. Today, however, Samet campaigns against ozone as if he had never written that paper.

After a two-part science and legal critique that I wrote on Samet's 2000 New England Journal of Medicine 20-city study of effects of air pollution at the website of the American Council on Science and Health, 5,6 James Enstrom, Ph.D., research professor at the University of California at Los Angeles, contacted me and asked for assistance with his efforts to stop California government efforts to create more air pollution regulations that would harm business and industry. I submitted public comments opposing proposed EPA particulate and ozone regulations in 20067 and 2007, 8 with no effect on EPA policy or attitude. EPA continued to make absurd claims that this or that air pollution regulation would save lives.

During that same period, I benefited from the statistics expertise of S. Stanley Young, Ph.D., of the National Institute for Statistical Science in Research Triangle Park, North Carolina.

U.S. EPA Board of Scientific Counselors

In 2007 Enstrom, Young, and I decided to approach EPA's Board of Scientific Counselors (BOSC), an outside independent scientific advisory group that was supposed to monitor and critique EPA science and policy making to encourage research compliance with basic scientific rules. BOSC was composed of members of high professional standing who were in private or state activities, and not EPA employees.

We articulated our positions, based on our areas of concern for BOSC subcommittee meetings in late 2007, and then the executive committee in early 2008. Our pleas and arguments were:

1) Irresponsible and false epidemiology and toxicology by EPA researchers claimed an effect that clearly fell well below any threshold needed to show a toxic effect in observational epidemiological population studies. Evidence for claimed air pollution death effects was inadequate to prove any causation and was asserted without a plausible toxicological mechanism.

2) Studies with multiple inquiries exaggerate the chance of false positives. The EPA was misusing the concept of statistical significance by failing to adjust for the multiple inquiries.

3) The EPA and its sponsored researchers and reviewers ignored studies that disproved their theories and suffered from tunnel vision and confirmation bias. Moreover they persecuted researchers like Enstrom who found results that didn't support the EPA agenda.9

I traveled to Maryland to present my concerns in person to the BOSC subcommittee of the Human Health Risk Assessment Committee, and Enstrom and Young presented by telephone. After waiting through hours of presentations by insider EPA officials and researchers before the scheduled public comment period, each of us was allowed only three minutes. Considering the inhospitable reception we received, it was not surprising we were the only outside commenters. Many lectures of an hour or more had been followed by laudatory comments from other EPA employees and officials present. I also noted that the roster of

committee members was clearly made up of people who had previously, or would in the future, want to be grantees of EPA largesse. It was definitely a home game, with home umpires.

I reviewed the Board of Counselors minutes for the previous five years and found there were no public comments at Board of Counselors meetings in those years. Even highly placed people in private industry, who were severely affected by its regulations, had no taste for criticizing EPA or its sponsored researchers. Favoritism and influence peddling are constant factors in governmental programs. Enstrom, Young, and I decided that appeals to the supposedly independent BOSC were worthless. Nonetheless, we made presentations to another subcommittee and then the BOSC executive committee.

The CARB Toxic Air Machine Project of 2007-2008

The battle was over at EPA, since it was a fixed game, but at the same time there was a battle going on in California led by Enstrom, which heated up in 2008 because of a new set of diesel engine rules focused on fine-particulate air pollution. These regulations were proposed and supported by research sponsored by EPA and the California Air Resources Board (CARB), a subdivision of the California EPA.

In 2005 Enstrom published his results of a robust and current study on the effects of fine-particulate air pollution in California. The study (10) involved 50,000 people in the years 1973-2002. It showed no premature death effect in California from fine- particulate air pollution. Moreover, California's air pollution of the 1950s and 1960s had declined for 30 years. Nonetheless, the increasing rate of asthma was misrepresented as a sign of an air pollution crisis justifying more air pollution regulations for no discernible benefit. Enstrom was also concerned that economic hardships would prove to be important causes of deprivation and decreased human life expectancy, as demonstrated in reliable population studies.11

In 2007, the CARB "solicitation" and review process was set up for a document entitled "Methodology for Estimating Premature Deaths Associated with Long-term Exposure to Fine Airborne Particulate Matter in California." The process included three scientific advisors and six "independent" but paid reviewers well known to, and allies of, CARB. Then CARB staff in May of 2008 released a draft report and proposed regulatory regime, claiming that air pollution caused premature deaths in California. A public comment period began, and the CARB business-as-usual process ran into vigorous critiques 12 submitted by Enstrom and other distinguished public health scientists and engineers in July 2008.

Public criticisms of the CARB draft report included:

1) Panel reviewers were reviewing their own or their close colleagues' air pollution studies.

2) CARB had discarded the Enstrom study and ignored geographic and time trend evidence available in the reviewed research that argued against their conclusions of air pollution death effects in California and the need for more regulations.

3) CARB had failed to adjust for changes in engines and emissions that also made older studies invalid.

4) Basic rules of the sciences of epidemiology and toxicology were violated in the CARB research that made claims based on small associations that were inadequate to claim a premature death effect.

My critique 10, pp 129-135 of the comments document discusses basic principles of scientific evidence that the EPA violates in its overreach. According to the Federal Judicial Center's Reference Manual on Scientific Evidence, 13, 14 which discusses the magnitude of toxic effect required in observational studies that are

used in public health toxicology research, an agent was more likely than not the cause of an individual's disease when the relative risk (RR) is 2.0, that is, a 100 percent increase in the disease or effect (e.g. premature death) in the exposed population. For example, the research on effects of cigarette smoking showed the RR of lung cancer in cigarette smokers is 10.

An RR greater than 2.0 would permit an inference that an individual plaintiff's disease was more likely than not caused by the implicated agent. None of the cited foundational and supportive studies EPA or CARB use to justify air pollution regulatory regimes have the minimum RR of 2 needed to assert evidence in associations of causation.

While epidemiologists study population effects, toxicologists study adverse effects. In the early 1950s, Sir Austin Bradford Hill, British icon of public health research, originated nine criteria referred to by the Federal Judicial Center in the Reference Manual for proving toxicity. Hill's first and most important criterion was evidence of a measurable and significant toxic effect. Other criteria include that the toxic effect proposed has to be plausible, has to make temporal and dosage exposure sense, and should be evaluated to make sure some other factor is not in play.15

EPA has consistently disregarded the Bradford Hill criteria, in particular using small associations that fail the test of adequate evidence of effect. There is no real knowledge of actual exposure of individuals alleged to be affected or dead, and certainly no assurance that outside air quality is the exposure that is appropriate to measure, since people spend the majority of their time indoors. A final and important consideration is that EPA research shows no evidence of a current understanding of a plausible mechanism for fine-particle toxicity or lethality.

CARB staff in October 2008 issued a final report that was the same as the preliminary draft report of May 2008. CARB staff admitted that they didn't show the public scientific critiques to the expert panel or request an expert response to those criticisms of CARB research conclusions or policy proposals.

In December 2008, Enstrom and three other prominent California air pollution experts directly contacted CARB board members to urge rejection of the 2008 report. The four also wrote a public letter to CARB to recommend that CARB reassess the report and delay any decision on air pollution and diesel regulations.16

Enstrom and Young checked the credentials of Hien Tran, lead author of the CARB Report on Fine Particles and Premature Death in California, and found that he had a fake Ph.D., purchased for $1,000 from a drop box, Thornhill University.17 Enstrom and others also pursued another scandal—that CARB executive Mary Nichols knew of the Tran fraud and had not reported it to the CARB Board before Dec 12, 2008, when it voted to approve the Truck and Bus Regulation. Enstrom's research into the enabling legislation for CARB also found that most members of the Scientific Review Panel on Toxic Air Contaminants had served in their positions longer than the specified term of 3 years without following the nomination and appointment process of members required by the 1983 enabling statute. Pacific Legal Foundation filed a lawsuit in June 2009 to force compliance with the nomination and appointment process, resulting in the removal of five of the nine members.

A taxpayers' protest was held with speeches and demonstrations at the State Capitol on Aug 28, 2009, reinforced by the sound of a 220-truck convoy sponsored by the California Dump Truck Owners Association (now the California Construction Trucking Association). The convoy circled the Capitol building and, on

cue, sounded truck horns for one minute. The convoy and the Capitol steps rally on California agency overreach were not covered by the press, but the legislators were there.

Business leaders and industry sectors that use diesel engines raised their voices. Dr.Bill Wattenberg, an engineer and influential talk show host from San Francisco's KGO, railed against CARB. Bloggers and other radio hosts joined in. Bryan Bloom, Lee Brown, and Betty Plowman and other trucking industry people were eloquent in public meetings. Jay McKeenan for the California Independent Oil Marketers Association, representatives of the logging industry organizations, Bill Davis with the Southern California Contractors Association, and Shelly Sullivan of the California Manufacturers and Technology Association, all pressed for a CARB suspension of the new diesel rules and a sensible agency retreat from its aggressive stance. Skip Brown, construction executive, was a steady and important participant as a speaker and writer.

California Assemblyman Roger Niello (R-5th Assembly District) presented a bipartisan letter with 52 signers demanding that CARB suspend the new diesel rules. Senator Robert Dutton (R-31st Senate District) and Assemblyman Dan Logue (R-3rd Assembly District) introduced bills to slow down CARB implementation plans on greenhouse gas and global warming regulations. Gov. Arnold Schwarzenegger weighed in to advocate a suspension of any new fine particulate/diesel regulations until the California economy could recover.

As a result of this 2-year campaign, CARB attempted to repair its damaged reputation for reliable research with a full-day scientific discussion and "cage match" debate on Feb 26, 2010 at the California EPA hearing room in Sacramento.

CARB designated three experts from the original scientific review panel: Daniel Krewski, Ph.D., Michael Jerrett, Ph.D., and Arden Pope, Ph.D., well-credentialed and also longtime friends and beneficiaries of CARB and EPA grants, members of the insider air pollution club with senior status. CARB paid for them to appear just as they had paid for previous research and review work.

Krewski has headed a large group that did a national study.18 A close look at the results showed that they found no air pollution "associations" that would support a claim of human health effects in California, but they ignored their own results, which would argue against their basic premise. During the symposium, Jerrett admitted that he couldn't find an air pollution health effect in California, but a year later he manipulated the data to show a minor association in one of his models19 created by a trick in methodology and geographic gerrymandering that he called "conurbation."20 As noted above, the Pope and Dockery group 3,4 have been prolific and always predictably produced studies with very weak associations that they claim support their position that air pollution kills.

For the opposing public critics, James Enstrom, Ph.D., Fred Lipfert, Ph.D., Robert Phalen, Ph.D., Roger McClellan, D.V.M., Suresh Moolgavkar, M.D., Ph.D, and Tom Hesterberg, Ph.D., M.B.A., appeared. These well-qualified researchers urged no more regulations and no more exaggeration of the science on air pollution health effects.

The webcast is seven hours long. 21 The net effect was that the public commenters exposed the nature of the CARB malfeasance on human health effects science, and demonstrated that the CARB research project was a setup that involved conflicts of interest and a failure to objectively evaluate competing data and evidence on the question of California air quality and its effect on health.

No regulatory relief came from the debate and the proof of CARB malfeasance, and CARB proceeded with the originally planned air pollution regulations.

Washington Politics

The Space Science and Technology Committee of the House of Representatives contacted me in 2010, and I provided information from the CARB wars and the previous challenges of EPA air pollution research claims and policy making. Congress had hearings in the fall of 2010 and through 2011 on EPA air pollution research and regulations. In 2011 and 2012, the House Energy and Commerce Committee also had activities and an interest, and in February 2012 former chairman Rep. Joe Barton (R-Texas) gave a speech outlining the perfidy of the EPA on many aspects of science and policy, as well as legal aspects of EPA misconduct.

Barton condemned:

- EPA's refusal to assess risk and benefit on regulations;
- EPA's burdensome and nonsensical power plant regulations;
- EPA's failure to cooperate with congressional oversight;
- Persistent and flagrant conflicts of interest among EPA researchers and advisers who receive tens of millions of dollars in research grants from the agency while serving as reviewers of EPA research;22
- EPA researchers' refusal to comply with basic rules of public health research in toxicology and epidemiology;
- Inappropriate reliance on the precautionary principle;
- Circumvention of congressional oversight; and
- Grant-giving to non-governmental advocacy groups that then enter into collusive lawsuits and aggressive regulatory requests that promote the agency's agenda and expand its regulatory and political power.

As Barton pointed out, "I believe that the American public and taxpayers should not be paying for an agency that manipulates data and funds researchers in the form of exterior grants, who in turn serve on the internal committees within the EPA to create policy and work in an oversight capacity. This is an incredible conflict of interest to the American public."23

Rep. Barton's dressing-down of EPA and its administrator was a first step in the right direction. But now Rep. Barton and his colleagues need to follow through by implementing real solutions that will stop EPA's regulatory excesses.

EPA and the Admissibility of Scientific Evidence

EPA research on human health effects of air pollution consistently violates the rules of science and is not admissible in a federal court under the rules of Daubert v. Merrell Dow, 509 U.S. 579 (1993). The Daubert majority opinion, written by Justice Harry Blackmun, discarded the old rule of "generally accepted" for scientific testimony and evidence, from the 1923 case of Frye v. United States, 293 F. 1013 (D.C. Cir. 1923) and adopted new, more rigorous tests for admissibility of science testimony and evidence, under Federal

Rules of Evidence (1975), particularly Rule of Evidence 702 on Testimony by Experts. The rule provides that if scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue (Rule 104 test), a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise.

In his written opinion, Justice Blackmun provided an erudite discussion on the philosophy of science, with a strong dose of the theories of a respected philosopher of science, Karl Popper.

Justice Blackmun's major points were as follows:

1) Trial judges were the gate keepers to assure that reliable science was admitted as evidence.

2) Scientific testimony and other scientific evidence had to be consistent with everyday good scientific practice.

3) The science would be assessed generally as follows:

a. The general acceptance rule of Frye did not survive the new Federal Rules of Evidence.

b. Knowledge is more than subjective belief or unsupported speculation; it must be supported by evidence and proven methods.

c. An expert witness is permitted wide latitude under the federal rules of evidence to offer opinions, including those that are not based on firsthand knowledge or observation.

d. Under Federal Rule of Evidence 104, a federal trial judge must determine the threshold question of whether the evidence is relevant and material to the case and will assist the trier of fact.

Justice Blackmun continued that if the threshold test of Rule 104 is satisfied (3d above), then the judge, in applying the rules of Daubert, must assess the admissibility of the scientific evidence and testimony on the basis of four tests under Federal Rule of Evidence 702 on Testimony of Experts:

1) Whether the theory or technique can be and has been tested;

2) Whether the theory or technique has been subjected to peer review and publication (this test is not dispositive, only additive);

3) Whether the technique or method has a known or potential rate of error; and

4) Acceptance of the theory or technique within a relevant scientific community of scholars.

Professor Michael Fenner of Creighton Law School wrote a helpful, in-depth review of the Daubert opinion. 24 In *Judging Science*, 25 Kenneth Foster and Peter Huber (MIT Press 1995) also review and analyze Daubert, providing much background analysis on the problems of junk science and fallacious science and also on the methods that produce reliable evidence and avoid scientific negligence and misconduct.

The Federal Rules of Evidence provide a means to challenge EPA-sponsored research, claims, conduct, actions, and policy-making. The burden of the challenge to an action, or ruling or fine or penalty, is to prove that the agency was arbitrary and capricious in its analysis of the pertinent science and research on human health effects and detriment. A common-sense understanding of those words entails actions taken without good justification or rationale. The courts have been inclined to be excessively deferential and allow agency

hegemony, even refusing to hear arguments on the arbitrary and capricious standard for agency acceptance of scientific research assertions.

Jurisprudence allows for judicial deference to agency discretion in matters of ambiguous statutory provisions, described by Justice Antonin Scalia in *Whitman v. American Trucking Association*. 26 What the erudite Justice Scalia fails to constrain is the inordinate and inappropriate expansion of the deference allowed EPA in reference to interpretation of ambiguous statutory language to include arbitrary and capricious agency acceptance of what would be arguably inadmissible scientific testimony and evidence.

Judges are, however, and always have been, the ones to decide what's admissible as evidence. Agency discretion under the jurisprudence of the Chevron decision 27 should not allow unreliable scientific evidence into the record under the rules of *Daubert*, whether it's a hearing or a trial. The evidence must be admissible for purposes of proving that the agency is or is not being arbitrary or capricious, which makes the decision on evidentiary admissibility and reliability separate from whatever idea the court might have about agency authority and discretion.

Unreliable scientific evidence is inadmissible and therefore cannot be used to justify agency actions. The admissibility rulings on evidence trump some arcane idea about agency discretion that is all tied up in the jurisprudence on congressional delegation. There is no law that Congress has passed that permits agencies to use and promote junk science.

In the excessive support of congressional delegation to agencies under the statutes, and the general deference for agency discretion under Chevron, Scalia allows EPA research to cheat and avoid a challenge under the "arbitrary and capricious" standard. Justice Scalia just plain ignores the commonly and legally understood meaning of "arbitrary and capricious." Proposing inadmissible scientific evidence and testimony on critical research assertions that are foundations for policy and regulatory action would certainly cross the threshold of "arbitrary and capricious" under the Administrative Procedure Act.

The Role of the Administrative Procedure Act (APA)

The Administrative Procedure Act (APA) allows a successful challenge of agency conduct when that action is arbitrary (without good reason) and capricious (on a whim and without a good reason). Violating scientific rules, like the ones that are clearly outlined in the *Reference Manual on Scientific Evidence* 11, 12 to educate judges on science, would certainly raise the question of irrationality that is the fundamental issue for claiming that an agency has acted in an arbitrary and capricious manner.

The courts have, however, been very lenient with the EPA on the violations of scientific rules and provided many opportunities for agencies to violate the rules of science, so legislative actions may be necessary to force better science and policymaking at EPA. The alternative is to find a judge with integrity and an appellate court that doesn't undermine a judgment of inadmissibility, or will entertain and find valid an appeal to reverse an improper judgment on Daubert admissibility.

Legislative Remedies

In the political sphere, Congress can modify standards of administrative and judicial review to demand good science and a better standard for agency conduct, with more reasonable rules on challenges to EPA actions. This is similar to the rules for challenges to actions by the Occupational Safety and Health Administration, which carry a preponderance-of-evidence burden.

The pertinent legislative act is the Congressional Review Act (CRA), found at 5 U.S.C. 801, which allows Congress to jump in when the agencies are involved in misconduct. CRA was enacted as section 251 of the Contract with America

Advancement Act of 1996, also known as the Small Business Regulatory Enforcement Fairness Act of 1996 (SBREFA). The law allows Congress to review, by means of an expedited legislative process, new federal regulations and, by passage of a joint resolution, to overrule a regulation.

Another legislative effort to bring the pressure to bear on the federal agency and their sponsored researchers is the **Data Quality Act**, which requires agency-sponsored research to hold to good scientific principles or be subject to review and possible modification or recision.

Even without legislation, responsible, competent, and serious legislators can find reasons to question EPA conduct, and lawyers can frame evidentiary challenges so that the courts and administrative hearings will be required to make clear rulings on admissibility of scientific evidence with an accompanying rationale for appellate review.

A bad evidentiary ruling is a reversible error; a good ruling will nurture good science in the courtroom. No lawyer but a pettifogger would admit to arguing for bad science that violates the public trust.

At present EPA, following Samet, 28 asserts the theory of "no threshold" for a toxic effect of air pollution, allowing EPA to pursue any pollutant to the last molecule. This impossible goal allows for unlimited expansion of EPA power. Chemical toxicology still is based on thresholds. **"No threshold" chemical air pollutant toxicology turns the Clean Air Act (42 USC 7401. 1963, amended 1970, 1990) on its head and nullifies and abandons the strategy Congress intended.**

Human Experimentation Scandal

As previously described in this journal, 29 EPA has been sponsoring research in which human subjects are exposed to air pollutants at levels far exceeding those EPA declares to be toxic or lethal. It is illegal, unethical, and immoral to expose experimental subjects to harmful or lethal toxins.30 The Reference Manual on Scientific Evidence, 3rd ed. (2011), [12, p 555] declares that exposing human subjects to toxic substances is "proscribed" by law, and cites case law. The editor of Environmental Health Perspectives (EHP) refused a request by Steve Milloy of JunkScience.com to withdraw a paper based on one such study and conduct an investigation.31

According to information obtained by Milloy from a Freedom of Information Act (FOIA) request, a University of North Carolina research study exposed 42 people to what EPA says are harmful or lethal levels of fine particles, with some receiving 10 times EPA's declared safe level of 35 micrograms per cubic meter of air. The EPA human experiments described were conducted from January 2010 to June 2011, and ended three months before then-EPA Director Lisa Jackson's congressional testimony, during which she still asserted dramatic claims of the lethality of small particulates less than 2.5 microns in diameter (PM2.5), claiming thousands of deaths and hundreds of billions of dollars in economic consequences from the deaths and disabilities caused by fine particles.

There have been no publications of toxic effects as declared by the authors of the paper, other than the one case report of a cardiac arrhythmia described earlier; 29 the researchers failed to report that none of the other

subjects had any adverse effects, despite the obligation of researchers to report results both for and against their hypothesis.

Did EPA risk the deaths of 42 subjects? Or are EPA officials lying in their testimony about the dangers of small-particle air pollution and deliberately misleading Congress and the public?

After filing complaints with EPA officials and the editor of EHP, Milloy and I filed complaints with the North Carolina Board of Medicine and the University of North Carolina (UNC) School of Medicine. The North Carolina medical board found no violation of the Medical Practice Act by the physicians, and no action was taken by the UNC School of Medicine.

A lawsuit was filed in Federal District Court in Arlington, Va., to ask for injunctive relief or a remedy that would stop the human experiments. The Court said it didn't have the authority or jurisdiction to stop the human experiments, but declarations under penalty of perjury obtained from officials of the EPA research team at UNC Chapel Hill School of Medicine were revealing.

Eugene Cascio, M.D., a lead EPA physician in the research team, declared that 10 domestic medical schools and six foreign medical schools were doing human exposure experiments. They included some of the most prominent medical schools in the United States—Rutgers, Rochester, Ohio State, University of Michigan, Michigan State, University of Washington, University of California at Los Angeles, University of Southern California, and Lovelace Clinic affiliated with the University of New Mexico. The foreign medical schools included three in Europe, one in Canada, and two in the UK.32

Two other declarations produced by EPA officials in the lawsuit were critical to understanding EPA misconduct. Martin Case, program administrator, declared that he told the subjects they could die from the exposures, but he did not write that warning in the consents obtained.33 Milloy has obtained the consent forms from UNC and other medical schools involved in the project for human experimentation, and none of programs warned subjects of EPA's position that fine particles were toxic, lethal, and carcinogenic, and that the subjects might suffer the consequences.34

Robert Devlin, Ph.D., senior research official for EPA and part of the UNC team, stated in his declaration under penalty of perjury that the EPA was sponsoring the human experimentation because the results of epidemiological studies are not reliable enough and do not establish a strong enough case for toxicity of air pollution.35

In paragraph 8, Devlin states:

> Controlled human exposure studies conducted by EPA scientists and EPA-funded scientists at multiple U.S. universities fill an information gap that cannot be filled by large population studies. In 1998 the Committee on Research Priorities for Airborne Particulate Matter was established by the National Research Council in response to a request from Congress. The committee was charged with producing four reports over a five- year period which describe a conceptual framework for an integrated national program of particulate-matter research, and identified the most critical research needs linked to key policy-related scientific uncertainties.

The committee states on page 36 of its report:

> Controlled human exposure studies offer the opportunity to study small numbers of human subjects under carefully controlled exposure conditions and gain valuable insights into both the relative deposition of inhaled particles and the resulting health effects. Individuals studied can range from healthy people to individuals with cardiac or respiratory diseases of varying degrees of severity. In all cases, the specific protocols defining the subjects, the exposure conditions, and the evaluation procedures must be reviewed and approved by institutional review boards providing oversight for human experimentation. The exposure atmospheres studied vary, ranging from well-defined, single- component aerosols (such as black carbon or sulfuric acid) to atmospheres produced by recently developed particle concentrators, which concentrate the particles present in ambient air. The concentrations of particles studied are limited by ethical considerations and by concern for the range of concentrations, from the experimental setting to typical ambient concentration, over which findings need to be extrapolated.

Controlled human exposures studies have been conducted for decades on important pollutants such as ozone, particulate matter, nitrogen dioxide (NO2), sulfur dioxide (SO2), VOCs [volatile organic compounds] emitted [in] new homes, and carbon monoxide (CO).

In paragraph 9 of his Declaration, Devlin states: "Controlled human exposure studies assess the biological plausibility of the associations observed in the large-population epidemiological studies."

So we have come full circle. For 20 years I have argued that EPA is involved in corrupted, invalid, unreliable epidemiology. Now, under pressure from a lawsuit for unethical conduct, it admits what we knew already, that epidemiology is being misused as a false portfolio of evidence of air pollution toxicity. The most astounding aspect of this human experiments scandal is the refusal of state boards of medicine, institutional review boards (IRBs), deans of medical schools, and EPA officials to investigate and stop the misconduct. This is in spite of the well-known and remembered Tuskegee and horrific wartime

Nazi/Japanese medical experiments on prisoners.

What we have discovered with EPA misconduct and that of the grantees at numerous medical schools is very sobering. These are not trivial violations of the ethical rules on human experimentation with which the IRBs are familiar. The rule is that one cannot perform harmful human exposure experiments— period. In only a very few circumstances where significant benefit is anticipated could subjects be exposed to harmful substances, after they are informed of the risks.

Conclusion

For 20 years or more EPA has promulgated bad epidemiology and bad toxicology that eventually evolved into research with unethical human exposure experiments. There is no easy way to excuse unethical human experiments to substantiate claims made in congressional hearings, despite lack of evidence, that air pollution or other forms of pollution are toxic and lethal.

If EPA is lying about the toxicity, the regulations fall. If it isn't, a federal agency is committing battery and unethical research that is criminal, unethical, and violates agency rules on human research. Either way, innocent experimental subjects are victimized.

Daubert and the *Reference Manual* guidelines could be used to restore sanity and objectivity to EPA regulatory activities so that they would improve public health policy-making rather than serving a political agenda.

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7. Dunn and Milloy on EPA sponsored Human Experiments using small particles emissions.

Environmental Protection Agency's Air Pollution Research: Unethical and Illegal?

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- www.jpands.org/vol17no4/dunn.pdf

"First, do no harm"is a fundamental precept of medical ethics. So how do U.S. Environmental Protection Agency physicians explain their non-therapeutic experiments in which they exposed health-impaired people to high levels of concentrated diesel exhaust and other air pollutants?

A federal court may soon help clarify this dilemma.

Since at least 2004, EPA physicians have been intentionally exposing human beings to various forms of concentrated airborne particulate matter (PM), including diesel exhaust, at an EPA laboratory at the University of North Carolina School of Medicine (UNC). The diesel exhaust is generated by idling a diesel truck with its exhaust pipe located right under the air intake for the exposure chamber.

The university not only houses the EPA facility, but also provides on a contract basis the mandatory institutional review board (IRB) intended to serve as the last line of defense for human study subjects.

Although these experiments materially violate every law, regulation, and standard developed since World War II for the protection of human subjects, there are two primary violations.1

First, these experiments should never have been approved by UNC or conducted by EPA given the allegedly lethal nature of PM as determined by EPA.

Since 1997, the agency has regulated PM on the basis that it kills people. In 2004, EPA clarified its views of PM's lethality by concluding that any inhalation of PM could result in death within hours of exposure.2 The EPA reiterated this view in its 2009 scientific assessment of PM.3

In July 2011, Dr. Jon Samet, chairman of EPA's Clean Air Scientific Advisory Committee, wrote in the New England Journal of Medicine that there is no safe exposure to PM.4 This view was repeatedly echoed by EPA air chief Gina McCarthy in a February 2012 letter to House Energy and Commerce Chairman Fred Upton (R-Mich.).5

EPA Administrator Lisa Jackson testified before Congress in September 2011: "Particulate matter causes premature death. It doesn't make you sick. It's directly causal to dying sooner than you should. She added,"If we could reduce particulate matter to levels that are healthy we would have an identical impact to finding a cure for cancer."6 Cancer kills about 570,000 in the U.S. annually, according to the American Cancer Society.

In addition to the EPA-determined lethal nature of PM, EPA also says there is strong evidence that PM is carcinogenic.7

These characterizations of PM essentially portray it as one of the most toxic substances known to man—at least according to EPA. Though every poison has a lethal dose, any exposure to PM can kill, and kill quickly (within hours), EPA claims. Although exposure to carcinogens like asbestos, benzene, and vinyl chloride may cause cancers decades after exposure, or after decades of exposure, these risks obviously pale in comparison to that of PM in the view of EPA.

EPA, then, is experimenting on human beings with what it views as one of the most toxic substances known to man for the simple (and illegal) purpose of evaluating what would happen, apparently in an effort to bolster its epidemiological (i.e. statistical) claims.8,9 Worse, many of the study subjects are health-impaired, suffering from metabolic syndrome, asthma, old age, or combinations thereof.

The idea of a government agency deliberately exposing sick people to what it portrays as an extremely toxic substance is shocking. This is, however, only part of the story.

Second, informed consent is the cornerstone of medical practice and human testing protocols. Failure to obtain informed consent, among other misconduct, resulted in the execution of 16 of 23 Nazi doctors at the Nuremberg tribunal. The so-called "Common Rule" has been adopted by American medical researchers, including EPA, as a standard for conducting human experiments, and it prohibits harmful human experiments.10

Although EPA went through the motions of having its study subjects read and sign consent forms, the forms never mentioned that any exposure to PM could result in death within hours of the experiment. Study subjects were instead told, for example, "You may experience some minor degree of airway irritation, cough or shortness of breath or wheezing. These symptoms typically disappear two to four hours after exposure, but may last longer for particularly sensitive people."10

At least hundreds, and possibly thousands of human subjects have been so experimented upon by EPA physicians or EPA- grantee physicians at universities around the country. These experiments continue even as these concerns have been pointed out to EPA in recent months.

Has anyone been harmed? At least one 58-year-old obese woman with a personal and family history of heart problems had her experiment terminated early when she developed atrial fibrillation/flutter. The case was reported, 11 and it was said to be "the first case report of cardiovascular disease after exposure to elevated concentrations of any air pollutant." The rhythm resolved spontaneously about 2 hours after termination of the exposure. The authors concluded: "The resolution of the arrhythmia with termination of the particle exposure further supports a causal relationship between the two."They made this strong inference even while acknowledging evidence of a high frequency of supraventricular ectopy prior to exposure, numerous preexisting risk factors, and the fact that an

electrophysiologic study 6 weeks later revealed a re-entrant circuit, which was ablated. The authors suggested a potential mechanism of "disruption of the normal cardiac autonomic control," without

acknowledging the confounding factor of a potential emotional reaction to being in a setting resembling a gas chamber and being the subject of an exposure to an inhaled air mixture in a lab.

Although EPA physicians attributed the subject's arrhythmia to her PM exposure, they nevertheless did not modify the consent forms for subsequent human test subjects to reflect this risk.

As a result, the American Tradition Institute, a nonprofit public policy group, has filed suit in federal court against the EPA seeking an end to this illegal experimentation (American Tradition Institute Environmental Law Center v. U.S. EPA, Case 1:12- cv-01066-AJT-TCB, U.S. District Court for the Eastern District of Virginia—Alexandria Division).

Complaints have been filed with the North Carolina Medical Board concerning three of the North Carolina-licensed EPA physicians involved in the illegal experimentation. This investigation continues. The University of North Carolina School of Medicine has announced an internal review.

Congress has gotten involved, too. Sen. Jim Inhofe (R-Okla.) has requested that the Senate Environment and Public Works Committee, the committee responsible for overseeing EPA, schedule hearings on the scandal. Spearheaded by Rep. Paul Broun,

M.D. (R-Ga.-10), the House Science Committee has requested that the EPA Office of Inspector General conduct an investigation.

The lawsuit has already produced a notable admission of sorts from an EPA employee. In his declaration,12 EPA Clinical Studies Coordinator Martin W. Case asserted that he verbally informs human subjects in an ongoing trial that, "There is the possibility you may die from this." In addition to the shocking nature of this"warning,"even if it were acceptable to risk the lives of human study subjects for the sake of science—and it's not—such a warning would need to be in writing, according to federal regulations.

It's clear that "first, do no harm" was not a high priority concern of EPA physicians involved in this shocking experimentation. EPA and UNC are now in defensive postures, and the medical community needs to hold them accountable. Given past outrages of medical science, like the Nazi experiments and the Tuskegee syphilis experiments to name just two, what will the medical, political, and legal communities do to stop this ongoing research sponsored by a United States federal agency and funded with taxpayer dollars?

Another possibility is that the EPA does not believe its own testimony to Congress, and that oppressive, costly regulations have been imposed on American industry on the basis of flawed epidemiologic studies, unwarranted extrapolations, and contrived estimates of benefits. The experiments may be designed to find a potential mechanism of harm, like the one suggested in the case report by Ghio et al.11 If so, the very purpose of the experiments is to cause harm to human beings in an effort to justify false testimony.

[Editor's Note: In a letter from the Environmental Protection Agency Office of Inspector General, dated October 22, 2012, Assistant Inspector General for Program Evaluation, Carolyn Copper, indicated the agency "plans to begin an evaluation of the Environmental Protection Agency's (EPA's) Research on Human Subjects...to determine whether EPA: 1) Obtained sufficient approval to expose subjects to specific levels of diesel exhaust emissions or concentrated airborne particles; 2) Obtained adequate informed consent from human study subjects before exposing them to diesel exhaust emissions or concentrated airborne particles; 3) Adequately addressed any adverse events that occurred, including notifying the University of

North Carolina at Chapel Hill's Institutional Review Board (IRB), the Human Studies Review Board, and the Human Subjects Research Review Official, revising consent forms as needed, and providing clinical follow- up in accordance with the approved protocol." See http://junksciencecom. files.wordpress.com/2012/11/new-assignment-memorandum-on-oig- evaluation-on-epas-research-onhuman-subjects.pdf] .

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8. ESSAYS and ARTICLES THAT EMPHASIZE THE NATURE OF US EPA SCIENTIFIC MISCONDUCT

Hyperlinks to essays on correcting EPA science Abuses

- Science and the Toxic Scare Machine
- The EPA's Faulty Science Can Be Stopped
- A Strategy to Stop EPA Science Abuse

Hyperlinks to essays on the EPA human experiments scandal and legal and administrative review of the conduct of EPA

- http://www.americanthinker.com/articles/2012/06/epas_unethical_air_pollution_experiments.html
- The EPA Uses Children (and Adults) as Guinea Pigs
- http://www.americanthinker.com/articles/2016/08/epa_whitewashes_illegal_human_experiments.html
- http://www.americanthinker.com/articles/2017/04/swamp_diving_the_epas_secret_human_experiment_regime.html

  National Research Council Human Experiments investigation panel
- EPA Whitewashes Illegal Human Experiments

Arnett review of EPA misconduct on air quality research 2012

Politicized science, Enstrom v. environmental activists – 17(4):118-119, 2012

http://www.jpands.org/vol17no4/arnett.pdf

Enstrom study on small particles in Dose Response

https://junkscience.com/2017/04/epidemiologist-accuses-prominent-epa-funded-researchers-of-deliberate-misrepresentation-on-key-air-pollution-studies/

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CA study of small particles and ozone effects in 2017 by Young, Smith, Lopiano

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JAMA DI small particles article 2017

https://junkscience.com/2018/02/dr-john-dunn-blasts-jama-over-harvard-pm2-5-fraud/

http://www.americanthinker.com/articles/2017/12/medical_journal_perpetrates_the_noble_lie_that_american_air_quality_kills_.html

9. 2018 Enstrom reviews and exposes EPA air quality epidemiological misconduct 2017

https://junkscience.com/2018/05/pope-fails-to-find-error-in-enstroms-2017-reanalysis-of-pope-1995-pm2-5-study/

The study below is a reanalysis of earlier studies relied on by the US EPA.

Dr. Enstrom shows that the studies not only show small associations that are proof of nothing, but in some cases reanalyzing the data shows Confidence Intervals that include Relative Risk of 1.0 so the studies failed in every way to show an effect. Dr. Enstrom also provides information on a systematic effort by journals to suppress his expose'.

James E. Enstrom, Original Article Fine Particulate Matter and Total Mortality in Cancer Prevention Study Cohort Reanalysis

Enstrom J. Fine particulate matter and total mortality in cancer prevention study cohort reanalysis. Dose-Response: January-March 2017 1-12 DOI: 10.1177/1559325817693345
https://www.ncbi.nlm.nih.gov/pubmed/28473741

Abstract

Background: In 1997 the US Environmental Protection Agency (EPA) established the National Ambient Air Quality Standard (NAAQS) for fine particulate matter ($PM_{2.5}$), largely because of its positive relationship to total mortality in the 1982 American Cancer Society Cancer Prevention Study (CPS II) cohort. Subsequently, EPA has used this relationship as the primary justification for many costly regulations, most recently the Clean Power Plan. An independent analysis of the CPS II data was conducted in order to test the validity of this relationship.

Methods: The original CPS II questionnaire data, including 1982 to 1988 mortality follow-up, were analyzed using Cox proportional hazards regression. Results were obtained for 292 277 participants in 85 counties with 1979-1983 EPA Inhalable Particulate Network $PM_{2.5}$ measurements, as well as for 212 370 participants in the 50 counties used in the original 1995 analysis.

Results: The 1982 to 1988 relative risk (RR) of death from all causes and 95% confidence interval adjusted for age, sex, race, education, and smoking status was 1.023 (0.997-1.049) for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ in 85 counties and 1.025 (0.990-1.061) in the 50 original counties. The fully adjusted RR was null in the western and eastern portions of the United States, including in areas with somewhat higher $PM_{2.5}$ levels, particularly 5 Ohio Valley states and California.

Conclusion: No significant relationship between $PM_{2.5}$ and total mortality in the CPS II cohort was found when the best available $PM_{2.5}$ data were used. The original 1995 analysis found a positive relationship by selective use of CPS II and $PM_{2.5}$ data. This independent analysis of underlying data raises serious doubts about the CPS II epidemiologic evidence supporting the $PM_{2.5}$ NAAQS. These findings provide strong justification for further independent analysis of the CPS II data.

James E. Enstrom

Here is another article in 2018 that describes Enstrom's efforts to expose US EPA air quality effects research misconduct.

http://www.jpands.org/vol23no1/enstrom.pdf

Scientific Distortions in Fine Particulate Matter Epidemiology
James E. Enstrom, Ph.D., M.P.H.

ABSTRACT
The theoretical prevention of premature deaths from the inhalation of fine particulate matter is being used by the U.S. Environmental Protection Agency (EPA) to justify the National Ambient Air Quality Standard (NAAQS) and multibillion dollar regulations across the U.S., including the EPA Clean Power Plan and the California Air Resources Board (CARB) Truck and Bus Regulation. The epidemiology is severely flawed. Fine particulates probably make no significant contribution to premature mortality in the U.S. The publication of null findings has been blocked or marginalized and studies claiming excess mortality need to be reassessed.

Basics of Fine Particulate Matter

Fine particulate matter (PM2.5) is defined by its size (≤2.5 μm diameter), not its composition. Major sources in the U.S. are forest fires, commercial and residential burning, and diesel engines. In California, a major source is China; on some days up to 30% of fine particulates had crossed the Pacific Ocean.
Of these invisible particles, the average adult in the U.S., based on actual 2015 exposure levels, would inhale about 1 gram in an 80-year lifespan, assuming that he breathes about 10,000 liters of air a day at rest. For comparison, the amount inhaled while smoking 100 cigarettes is about 4 grams.1

In 1997, the EPA established the NAAQS for PM2.5 as 15 μg/ m3. This was lowered to 12 μg/m3 in 2012. This standard has been largely justified on the basis of secret science epidemiology. These regulations are very powerful and impose huge costs on American businesses. The PM2.5 NAAQS, has been used to justify several multi-billion-dollar rules, such as the EPA Clean Power Plan and the CARB Truck and Bus Regulation.

Although a significant effect from such extremely low levels is on its face highly implausible, the stringent EPA regulations are justified primarily by a claim of preventing premature deaths, assuming a value of $10 million per statistical life saved. The controversy over the issue was brought to general attention in 2002 by Professor Robert Phalen.2

Epidemiology of Fine Particulate Matter

The EPA claim that PM2.5 causes "premature deaths" is based on epidemiologic cohort studies purporting to show that the relative risk (RR) for total mortality is slightly greater than 1.0 in U.S. populations exposed to higher levels of PM2.5. No etiologic mechanism has been established, and there is no experimental evidence that inhalation of 1 g or 5 g of PM2.5 can cause death. Weakly positive RRs do not prove causality. Major difficulties include: (1) geographic and temporal variation in PM2.5 mortality risk; (2) exaggeration of actual human exposure by PM2.5 monitors, which measure ambient outdoor levels

far from the subjects; and (3) confounding variables such as co-pollutants. Moreover, the key study relied on by EPA, the American Cancer Society (ACS) 1982 Cancer Prevention Study (CPS II)3 is seriously flawed.

Reanalysis of the American Cancer Society Cancer Prevention Study II (ACS CPS II)

CPS II began in 1982 and is similar to the original CPS I, which began in 1959. The seminal paper published by Pope et al. in 19953 was so controversial that the Health Effects Institute (HEI) sought applications from teams consisting of two to four epidemiologists, statisticians, and airpollution exposure experts to conduct a reanalysis, including "sensitivity analyses to test the robustness of the original findings and interpretations to alternative analytic approaches."4 The HEI Reanalysis published in 2000 did not complete the mandated sensitivity analysis to assess the effect of alternate data.5 HEI published a report in 2009,6 which extended the mortality follow-up of the study from 1989 to 2000, but it did not incorporate the EPA Inhalable Particulate Network (IPN) PM2.5 data7,8 that I had called to the authors' attention in my 2005 paper.9

In 2016 I was able to obtain access to data in an original 1982-1988 version of CPS II. The data had been previously inaccessible since 1995 despite a congressional subpoena and repeated requests by different agencies. I am the only independent scientist who has gained access to the individual level data in both CPS I and CPS II. I was able to reproduce the same key results as Pope et al. by doing exactly what the authors did in 1995.3 However, their results were sensitive to the PM2.5 data that they used and to their particular analysis.

HEI did not follow its own mandate to conduct a comprehensive reanalysis. In particular, their sensitivity analysis was not done properly. Of the 13 teams that submitted reanalysis applications, HEI selected a 31-member team based in Canada, headed by statistician Daniel Krewski. It included a geographer, Michael Jerrett, and another statistician, Richard Burnett, but only had one epidemiologist, Yue Chen. Chen's degree was from Shanghai Medical University, and he was not a coauthor on either the 2000 HEI report5 or the 2009 HEI report.6 Thus, to reanalyze a major U.S. epidemiological study, HEI used a Canadian team that had essentially no epidemiologist.

An early clue to the existence of problems is seen in Figure 21 in the 2000 HEI Reanalysis Report.5 (Figure 1 in this article.) This map shows that in 50 cities across the U.S. the level of PM2.5 mortality risk varies. Higher risks were found mainly in the Rust Belt or the Ohio Valley, and levels were actually reasonably low

in California and throughout most of the western part of the U.S. Beginning in 2002, I asked the head of HEI, Daniel Greenbaum, and its principal scientist, Aaron Cohen, to send me the underlying data for that map. For 16 years, they have consistently refused to reveal this data to me.

Fine Particles and Mortality Risk
Figure 1. PM2.5 Levels and Mortality Risk in the U.S. [Reprinted from 2000 HEI Reanalysis Report,5 with permission.]

Thus, using the HEI PM2.5 data of Pope et al., 3 there is a statistically significant slight increase in RR of 1.082. That means that if the PM2.5 level increases by 10 μg/m3, the risk of dying goes up by about 8%. But, using the IPN PM2.5 data, the effect is nonsignificant, RR = 1.025 (95% CI, 0.990-1.061). Note that if one divides the U.S. into the Ohio Valley (Indiana, Kentucky, Ohio, Pennsylvania, and West Virginia) and the rest of the country, the RR is indistinguishable from 1.0, no matter what PM2.5 data is used. Only by combining the Ohio Valley, which has both a higher mortality risk and a higher level of PM2.5, with the rest of the country can HEI show a statistically significant effect.

My reanalysis10 has been published online since Mar 28, 2017, and so far its validity has not been challenged. The selection of data by HEI was also very interesting, as seen in Table 2. There were actually 11 counties in California that were part of the IPN network, and the HEI analyses omitted 7 of the 11 counties for reasons the authors have not explained. HEI had data from 50 different cities, and the only ones they included from California were Fresno, Los Angeles, San Francisco, and San Jose (in Santa Clara County). Two other counties that represent the extremes in PM2.5 levels are highlighted in the table. The Pope 1995 paper3 was based primarily on these extremes. HEI had Albuquerque, N.M., at 9 μg/

My analysis of the CPS II data revealed that the county of residence of subjects could be approximated based on the ACS Division and Unit numbers. The CPS II data were collected by about 70,000 researchers, including myself, who enrolled 1.2 million subjects in Fall 1982. I performed an analysis comparable to the HEI Reanalysis, as shown in Table 1. The PM2.5 data labeled IPN in the table was published in EPA reports from the Inhalable Particulate Network (IPN) by David Hinton et al. in 19847 and 1986.8 Because of the evasions that I have experienced in attempting to obtain information from HEI, I took a closer look at the 2000 HEI Reanalysis Report and found it actually contains the data that I used, although in a mislabeled and somewhat altered form. I have designated that data as HEIDC, which is labeled PM2.5 DC in the 2000 Report. This data was indirectly referred to in a couple of places in the 2000 HEI report, although it was not analyzed.

m3, as the lowest value, and Huntington, W.V., at 34.4 μg/m3, as the highest value. This is curious because the data that comes from the IPN network actually shows different high and low values. In fact, there is no measurement in the IPN for Huntington, W.V., but rather for Wheeling, W.V., listed in the IPN column. From the table, both the low and the high values are in California, both of which omitted from the HEI analysis. The low value is 10.6 μg/m3 in Santa Barbara County, and the high value is 42.0 μg/m3 in Riverside County. The PM2.5 DC data that I found in the 2000 HEI Report appendix table, labeled HEIDC by me, had more than 50 cities, but only five of the 63 total cities were from California. The IPN network as a whole has about 85 cities. These major inconsistencies need to be addressed by these investigators. And so far, there is nothing but silence. This is only one of the issues that must be addressed if the investigators want to maintain any credibility.

Table 1. Enstrom Analyses of ACS CPS II Data Using Three Sources of PM2.5 Data

Table 2. Comparison of Data on PM2.5 and Mortality from Enstrom and HEI9

Relationship between PM2.5 and Mortality in California

Because of the Feb 26, 2010, conference in Sacramento, which I attended along with Professor Robert Phalen, other prominent scientists, and impacted business groups, we were able to get an analysis done by HEI that dealt with the California portion of the national CPS II results. The California data was partitioned out from the national analysis in the 2009 HEI Report.6 Based on the four HEI California counties shown in Table 2, the RR is about 0.9, significantly below 1.0, as shown in Table 3. This inverse relationship was reproduced using either the HEI data or the IPN data. Of course, this relationship cannot be etiologically correct, but it shows what can result from data omission and manipulation.

Table 3. Relative Risk for PM2.5 and Mortality in California Based on Four Counties

Table 4. PM2.5 and Total Mortality in Six California Cohorts Both my analysis and that by Thurston et al. on the NIH
AARP cohort,14 summarized in Table 5, show no effect nation- wide or in California.

There are actually six California cohorts that have been used to analyze the relationship between PM2.5 and total mortality, as shown in Table 4. The cohort that I initially used is labeled CA CPS I;9 the cohort used by Jerrett et al.11 is labeled CA CPS II. The Adventist Health Study of Smog (AHSMOG) was the original cohort study in California.12 There are also the California Teachers Cohort,10 the "West" portion of the Medicare Cohort Air Pollution Study (MCAPS),13 and the National Institutes of Health-American Association of Retired Persons (NIH AARP) cohort, which was published in 2016 by Thurston et al.14 The NIH AARP cohort is supposed to be an open access database, but is apparently currently controlled by Thurston. I have been able to get access to only the California portion of the data, and my analysis shows no effect in California. Averaging all six cohorts gives an RR of exactly 1.00, which means no relationship between PM2.5 and total mortality.
The lack of an effect in California might explain why Pope et al.3 omitted seven California cities from the national analysis. As Figure 1 shows, there is tremendous variation across the country. Yet the most severe regulations are in California, despite the clear absence of mortality risk there!

Table 5. Comparison of Enstrom and Thurston Analyses for
U.S. and California

An International Perspective on PM2.5

Despite the null effect shown by their own data and analyses, prominent advocates of drastic measures to reduce PM2.5 levels state in a major paper in the May 13, 2017, Lancet that ambient PM2.5 was the fifth-ranking mortality risk factor worldwide in 2015. Aaron J. Cohen, until recently HEI Principal Scientist, is the lead author, and Pope is a coauthor. The study is part of the World Health Organization (WHO) Global Burden of Disease (GBD) Project and was largely funded by HEI. The article claims that PM2.5 causes 4.2 million deaths annually worldwide, with 88,000 deaths in the U.S. (see Table 6). The mean PM2.5 level is

8.4 μg/m3 in the U.S. and 58.4 μg/m3 in China. Clearly, the PM2.5 level and premature deaths are low in the U.S. and high in China, India, and Africa.

Table 6. Global Deaths Attributed to PM 15

Agenda-driven Science

Since publishing my 2005 critique of the relationship between PM2.5 and total mortality9 and my 2017 critique,10 I have sent numerous requests to Pope, ACS, HEI, and others, inviting a rebuttal. I have received no response that confirms or refutes any of my analyses. It has, however, been incorrectly asserted that, "The study by Enstrom does not contribute to the larger body of evidence on the health effects of PM2.5." ACS has criticized me for having CPS II data that they have deliberately tried to keep secret. My invitations to authors and ACS officials to attend meetings, teleconferences, and symposia have simply been ignored. They even ignored an August 1, 2013, subpoena from the U.S. House Science, Space, and Technology Committee.

The control over air pollution research and assessments that is recognized by EPA is not based on special expertise in epidemiology. Pope, the self-proclaimed "world's leading expert on the effects of air pollution on health," is a professor of economics at Brigham Young University and holds a 1981 Ph.D. in agricultural economics from Iowa State University, where he studied the dynamics of crop yields. Michael Jerrett, who is one of the most prolific publishers and a member of the HEI reanalysis team, has a 1996 Ph.D. in geography from the University of Toronto, and no formal training in epidemiology. Aaron J. Cohen, until recently HEI's Principal Scientist, does hold a 1991 D.Sc. degree in epidemiology from Boston University, but he has badly misused the principles and standards of epidemiology. Although he supervised the 1998-2000 HEI Reanalysis Project, he has refused to clarify findings from this project and has refused to confirm or refute the findings in my 2017 CPS II reanalysis. It is very disturbing that ACS has allowed CPS II data to be used for more than 20 years for research that misuses the principles and standards of epidemiology and that has nothing significant to do with cancer.

The principal qualification for admission to the elite circle of influence appears to be dedication to the agenda of global controls on economic activity via air pollution regulations. The conclusion reached by researchers is apparently predetermined, as stated in the last paragraph of the GBD study on ambient air pollution: "As the experience in the U.S. suggests, changes in ambient PM2.5 associated with aggressive air quality management programmes, focused on major sources of air pollution including coal combustion, household burning of solid fuels, and road transport, can lead to increased life expectancy over short timeframes."15

What is the state of scientific integrity? It is very dangerous to one's career to criticize views backed by powerful interests, and I do it only because I believe current trends are anti- science and dangerous to our country. Simply being a passive observer is no longer acceptable.

To disclose my own background, I obtained a Ph.D. in physics in 1970, but I became an epidemiologist starting in 1973 in order to apply the rigorous principles of physics to observational epidemiology. I had a long career as a research professor and researcher at the UCLA School of Public Health. My research has examined the influence of environmental and lifestyle factors on mortality, and has on occasion reached politically incorrect conclusions. My research in air pollution epidemiology has been strongly influenced by Dr. Frederick Lipfert and Professor Robert Phalen. In February 2010 I was terminated from UCLA without warning and told that my "research is not aligned with the academic mission of the Department." In

February 2015 I settled a three-year federal whistleblower retaliation lawsuit against UCLA and my termination was reversed. My case and some of the issues related to my air pollution epidemiology research have been discussed in this journal.16

My background and publications, including rejections of my research, often without peer review, are documented on my website, www.scientificintegrityinstitute.org. I believe that major journals simply will not accept articles that challenge the established view. Moreover, authors of the papers promoting PM2.5 premature deaths omit null results, even their own. For example, Jerrett is the lead author of a 2007 study that shows no increased mortality associated with PM2.5 in the CPS II cohort if the results are divided into five time periods.17 Although researchers are paid millions of dollars, they're not under any obligation to address any of the concerns about their work. Those who disagree with the agenda are denied research funding.

We must prevent American science from following historical examples like that of Trofim Denisovich Lysenko. He was a phony plant geneticist, who gained the favor of Joseph Stalin because he didn't believe in Mendelian genetics. Lysenko's views controlled much of Soviet agriculture in the 1930s, 1940s, and 1950s, with devastating effect. False crop statistics were published, and dissenting scientists were purged. Nikolai Vavilov, a renowned plant geneticist, was imprisoned by Stalin and died of malnutrition.
Concerns about integrity in Western science are being raised. Richard Horton, editor of The Lancet, writes: "The case against science is straightforward: much of the scientific literature, perhaps half, may simply be untrue. Afflicted by studies with small sample sizes, tiny effects, invalid exploratory analyses, and flagrant conflicts of interest, together with an obsession for pursuing fashionable trends of dubious importance, science has taken a turn towards darkness."18

A U.S. House of Representatives bill called the Secret Science Reform Act was passed in 2014 and 2015 in order "to prohibit the Environmental Protection Agency from proposing, finalizing, and disseminating regulations or assessments based upon science that is not transparent or reproducible." The bill was revived in 2017 as the Honest and Open New EPA Science Treatment (HONEST) Act, labeled H.R. 1430, and was passed by the U.S. House of Representatives.
American science needs to guard against the heirs of Sinclair Lewis's protagonist in his 1927 novel Elmer Gantry, an itinerant preacher who is able to sell false religion to gullible people. We have prominent scientists who have successfully sold the notion that inhaling 1 g of invisible particles over an 80-year lifetime can cause premature death.

Conclusions

There is strong evidence from two large national cohorts that PM2.5 does not cause premature deaths in the US. There is strong evidence that this relationship has been falsified by EPA, the Health Effects Institute, and leading researchers for more than 20 years. Better oversight to assure scientific integrity, such as access to data, transparency, and consideration of opposing views, is imperative.

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10. Dunn on US EPA Linear No Threshold Misconduct 2018.

This is a paper by the submitter Dunn that is intended to be an abstract as a presentation to a conference of the American Nuclear Society and the Health Physics Society on the problem of Linear No Threshold toxicology. The Conference is scheduled for early October 2018.

AN ENVIRONMENTAL NOBLE LIE,
LINEAR NO-THRESHOLD Radiation Biophysics Toxicology,
IT NEEDS TO GO

John Dale Dunn MD JD
American Nuclear Society/Health Physics Society Conference
Sept 30-Oct 3, 2018
Pasco, Washington

Abstract
The United States Environmental Protection Agency (USEPA) is charged with identifying and mitigating environmental risks. This article will discuss US EPA misguided decision to use Linear No Threshold as the template for Radiation Biophysics and Toxicology.
The Health Physics Society (HPS) has stated that reliance on the LNT model "...tends to foment the public's fear of all types of radiation . . . reliance on the LNT model, especially at very low doses and dose rates, is inappropriate and can exaggerate the risk." (Kirner 2017) (Ring et al. 2017). The HPS also condemns "collective" (cumulative) dose as a measure of biological radiation risk.
One hit or linear no threshold (LNT) radiation biophysics makes no sense as a theory for carcinogenesis. Most cancer cell types are hyper/multiploid due to telomeric mitotic dysfunction, not mutations of genetic code. Carcinogenesis is also enabled by immune system failure to eliminate malignant cell lines. Both phenomena are associated with aging.

The US EPA acceptance of the assertions on LNT of Biological Effects of Atomic Radiation (BEAR), Biological Effects of Ionizing Radiation (BEIR) and National Academy of Science (NAS) committees, has been so irrational as to assume there is no safe level of ionizing radiation. Nonsense.
The LNT cancer theorists ignore protective biological processes, even hormetic, certainly no effect evidence of low level radiation. (Ulsh 2010; Sacks and Siegel 2017; Welsh et al. 2017), Scott 2017), acknowledged by the National Council on Radiation Protection and Measurements (NCRP) over 15 years ago (NCRP 2001). "These experimental observations are not compatible with a single hit mechanism. . . hypothesis." (Trott and Rosemann 2000)
The fruit fly research by Hermann Muller and Curt Stern founded the LNT model, but the research actually showed a threshold, misrepresented by Muller, a committed advocate of LNT (Siegel et al. 2015; Calabrese 2017a, 2017b). Muller was a deceitful, relentless advocate of LNT, and, as a Nobel Laureate, very influential. (Calabrese 2017c)
The American Association of Physicists in Medicine (AAPM) strongly objects to the LNT approach as creating harm from adverse attitudes about imaging procedures. They consider the risks at or below 50 mSv [5 rem] for single procedures or 100 mSv [10 rem] for multiple procedures not detectable.
The USEPA use of LNT causes harm with no evidence of worthwhile benefit. US EPA claims that LNT is "conservative" and "cautious," translated as adoption of the misbegotten precautionary principle. The Fukishima mitigation, for example, was excessive, harmful and expensive, applied at doses far below the range of any negative public health consequences (Siegel et al. 2017c; Welsh et al. 2017).

Conclusions
The US EPA has been irresponsible and unscientific in its application of the Linear No Threshold template for radiation biophysics and toxicology. US EPA risk management is unscientific, unreliable and unjustified, wrongly derived from high dose rate environments and bench experimentation. Rat and mouse studies with exposures at lethal levels have created a long list of "carcinogens" that are then part of the LNT toxicology deception. (Calabrese 2018)
Society has become so fearful of radiation and chemicals that unnecessary steps are taken, and other risks are accepted, compliance costs are tolerated and are pursued energetically and expensively in a risk management environment of zero tolerance.
From the 1979 Three Mile Island to Fukishima in 2011, radiation incidents impacting large areas repeatedly show potential, variable risk for the immediate plant area, but, for example, even the terrible Chernobyl explosion, a stunningly limited harm from radiation beyond that.
The Fukishima event caused no radiation-related deaths (UNSCEAR 2013b), however the scare and the evacuation increased mortality, particularly in the elderly (Nomura et al. 2013; Yasumura et al. 2013; Uchimura et al. 2014, Ichiseki 2013) and the evacuations were scientifically unethical as a risk management strategy (Akabayashi and Hayashi 2012).
Changes, long overdue, on the matter of LDDR radiation risk management must go forward with the knowledge that adverse health effects are not detectable and that radiation exposures have a no effect, a harmful threshold of effect and even a sweet spot where radiation produces hormetic beneficial effects. (Calabrese 2013, Scott, 2017)
The USEPA Scientific Advisory Board (SAB) properly recommended a "change in the agency culture, change in how the agency works, and increased support for scientists and managers in programs and regional offices responsible for science integration." (Swackhamer and Burke 2012)
The radiation biophysics and toxicological precautionary principle needs a retirement in favor of rational risk assessment and mitigation.

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Below is my abstract/monograph for a presentation to the Gulf Coast Geophysical Societies conference scheduled for late September of 2018. Here I summarize much of the research on human health impacts from warmer temperatures—that shows the benefits of warming. That debunks the catastrophic and ominous claims of the US EPA. There are certainly other reasons to object to US EPA clams that CO 2 is a pollutant and dangerous, but underlying those claims is their fraudulent and unsupported claim that warming would be deleterious to human health—when the opposite is true.

This if offered as just one exhibit that shows the US EPA has been irresponsible in its claims about the impact of CO2 rise and warming—there are other scientific research studies that show the claims about

11. Dunn on Global Warming and Climate Change EPA misconduct—the scam of making Carbon Dioxide a pollutant.

Warming is a Benefit to Humans and the Biosphere

John Dale Dunn MD JD

The Intergovernmental Panel on Climate Change (IPCC) predicts a global temperature increase of 3C or more by 2100, but other experts believe the best guess is 1C or less. We assert that increases in average temperature of the planet from the current 60 degrees F. will be beneficial to human health and the biosphere.

IPCC's alarms have led to widespread fear of the health effects of global warming (Schulte, 2008) and even political attack ads claiming people are dying of "carbon pollution" (WMC, 2015). These statements have no basis in scientific research and in fact and based on the evidence, warming will be a benefit to all living things. Carbon Dioxide that increases to even 1000 PPM will be beneficial to the biosphere and make the planet more hospitable and arable.

In fact, the litany of climate extremes postulated by the IPCC has been falsified by the actual record of climate measurements and observations. None of the environmental disasters, human displacements and disruptions predicted have come to pass during the past ten years, even as atmospheric carbon dioxide has continued to increase. We all know of the temperature "pause" that has accompanied an increase in atmospheric Carbon Dioxide.

In this document the benefits of fossil fuel use, and even warming, if it did occur, are explained in greater detail.

A warmer planet is beneficial to humanity as warmer temperatures lead to decreases in temperature-related mortality, premature deaths due to cardiovascular and respiratory disease, and stroke occurrences, and has little if any influence on vector-borne diseases such as malaria and dengue fever since vectors generally are not respectful of the definition of "tropical diseases.

Cool and colder temperatures kill while warmer temperatures are beneficial. It is troubling that, in the face of this evidence, environmentalists and politicians continue to frighten people with predictions of "killer heat waves" in a slightly warmer world. And yet, such claims are made. Severe heat waves are a weather phenomenon, not causally linked to average global temperature. Deaths from heat waves are most dramatic in areas with lack of adaptation—or general medical care for the disabled—who suffer from poor housing and medical problems that make them more susceptible.

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Global Warming and Mortality Rates

- Medical research confirms and explains why cooler, colder temperatures cause increased disease and death rates. Warmer temperatures are associated with health benefits and decreased deaths.

- Population studies around the world show that warmer temperatures lead to a net decrease in mortality worldwide, even in those areas described as tropical.

- Carbon dioxide (CO2) is invisible, odorless, nontoxic, and does not seriously affect human health until the CO2 content of the air reaches approximately 15,000 ppm, more than 37 times greater than the current concentration of atmospheric CO2 (Luft et al., 1974). There is no reason to be concerned about any direct adverse human health consequences of the ongoing rise in the air's CO2 content now or in the future,

currently at about 400 parts per million (0.04%) since even extreme model predictions by warming advocates are for less than 2000 parts per million (2%).

The Intergovernmental Panel on Climate Change (IPCC), however, sees looming health threats. The Summary for Policymakers of IPCC's Working Group II's report for the Fifth Assessment Report (AR5) identified eight "key risk factors" regarding the effect of climate change on human wellbeing, all of them allegedly "identified with high confidence" (IPCC, 2014, emphasis in original). They are:

i) Risk of death, injury, ill-health, or disrupted livelihoods in low-lying coastal zones and small island developing states and other small islands, due to storm surges, coastal flooding, and sea level rise. 37[RFC1-5]

ii) Risk of severe ill-health and disrupted livelihoods for large urban populations due to inland flooding in some regions. 38 [RFC 2 and 3]

iii) Systemic risks due to extreme weather events leading to breakdown of infrastructure networks and critical services such as electricity, water supply, and health and emergency services. 39 [RFC 2-4]

iv) Risk of mortality and morbidity during periods of extreme heat, particularly for vulnerable urban populations and those working outdoors in urban or rural areas. 40 [RFC 2 and 3]

v) Risk of food insecurity and the breakdown of food systems linked to warming, drought, flooding, and precipitation variability and extremes, particularly for poorer populations in urban and rural settings. 41 [RFC 2-4]

vi) Risk of loss of rural livelihoods and income due to insufficient access to drinking and irrigation water and reduced agricultural productivity, particularly for farmers and pastoralists with minimal capital in semi-arid regions. 42 [RFC 2 and 3]

vii) Risk of loss of marine and coastal ecosystems, biodiversity, and the ecosystem goods, functions, and services they provide for coastal livelihoods, especially for fishing communities in the tropics and the Arctic. 43 [RFC 1, 2, and 4]

viii) Risk of loss of terrestrial and inland water ecosystems, biodiversity, and the ecosystem goods, functions, and services they provide for livelihoods. 44 [RFC 1, 3, and 4]

There is no scientific basis for believing global temperatures will rise to levels high enough to bring about any of these risks. Indeed, there is sound scientific support for believing warming will be a net positive rather than negative.

Here, we summarize only research on the effects of rising global temperatures on human health and the medical literature shows warmer temperatures and a smaller difference between daily high and low temperatures that results from some rising temperatures as occurred during the twentieth and early twenty-first centuries, reduce mortality rates (the subject of this section) as well as illness and mortality due to cardiovascular and respiratory disease and stroke occurrence.

Similarly, the research is quite clear that climate has exerted only a minimal influence on recent trends in vector-borne diseases such as malaria, dengue fever, and tick-borne diseases. Other factors, many of them related to economic and technological setbacks or progress and not to weather, are far more important in determining the transmission and presence of these "tropical" diseases that are not so tropical at all.

Warmer Temperature Impacts on Human Health

- Warmer temperatures lead to a decrease in temperature-related mortality, including deaths associated with cardiovascular disease, respiratory disease, and strokes. The evidence of this benefit comes from research conducted in every major country of the world.

- In the United States the average person who died because of cold temperature exposure lost in excess of 10 years of potential life, whereas the average person who died because of extreme heat related event lost no more than a few days or weeks of life because heat has a greater effect on more seriously debilitated and ill persons.

- In the U.S., some 4,600 deaths are delayed each year as people move from cold northeastern states to warm southwestern states. Between 3 and 7% of the gains in longevity experienced over the past three decades was due simply to people moving to warmer states.

- Cold-related deaths are far more numerous than heat-related deaths in the United States and the world. Coronary (heart attack) and cerebral thrombosis (stroke) account for about half of all cold-related mortality, events that are directed related to blood vessel and blood viscosity effects of cool or cold environments.

- Global warming, if it did occur, even to the degree predicted in the extreme, will reduce the incidence of cardiovascular diseases related to low temperatures and wintry weather by a much greater degree than the warming might increase the incidence of deaths or illness attributable to heat. Heat illness primarily produces fluid and electrolyte disturbances, loss of core temperature control and organ dysfunction from dehydration, circulatory failure and heat caused stress, not clotting events.

- The heat wave deaths of 1995 in Chicago and 2003 in Europe are pointed to by advocates of the claim that heat stress deaths will increase with any warming that might occur, but a closer look at heat event death rates in some of the studies below show acclimation increased awareness have blunted any heat stress death increases. In the case of Chicago and Europe temps rose to over 100 but the availability of air conditioning and ventilation along with attention to the needs of elderly and disabled individuals was determined to be a major reason for heat deaths.

- The heat deaths that occur during severe heat events are the result of stress and inability to acclimate to maintain normal core temperature control and avoid dehydration. Acclimatization and proper attention to the vulnerable populations failed in Chicago in 1995 and Europe, particularly France in 2003, for example with hundreds of heat deaths in the former and 20,000 or more deaths in the later.

- A large body of scientific examination and research contradicts and disproves the claim that malaria will expand across the globe and intensify as a result of CO2-induced warming. Malaria is historically a disease that was endemic to cool and even cold climates like Finland and Russia but has been suppressed by hygienic and vector control

measures.
- Concerns over large increases in vector-borne diseases such as dengue as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue disease. The Aedes Aegypti Anopheles and Asian Tiger mosquitos all have been found at higher latitudes.
- While temperature and climate effect the geographical distribution of ticks, they are not among the significant factors determining the incidence of tick-borne diseases. Moreover the effect of small increases in climate temperature, if does occur with certainly not impact the range of ticks that now live in the high latitudes, even in the mountains of those high latitudes.

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Basis in Medical Science

Medical science explains why colder temperatures often cause diseases and sometimes fatalities whereas warmer temperatures are associated with health benefits.

Wang et al. collected daily mortality and meteorological data from 66 communities across China over the period 2006-2011. They then subjected these data to a series of analyses to elucidate the relationship between cold spell characteristics and human mortality. And what did those analyses reveal?

Not surprisingly, cold spells significantly increased human mortality risk in China. As indicated in Figure 1 below, the combined cumulative excess mortality risk (CER) for all of China when defining cold spells with a 5th and 2.5th percentile temperature intensity threshold was 28.5 and 39.7 percent, respectively. However, there were notable geographic differences; CER was tempered and near zero in the colder/higher latitudes, but increased to 58.7 and 92.9 percent at the corresponding 5th and 2.5th percentile temperature intensity

thresholds for the warmest and most southern latitude. Such geographic differences in mortality risk, according to the authors, are likely the product of better physiological and behavioral acclimatization of the northerly populations to cold weather.

Clearly, cold spells kill; and as has been found in almost every study of the subject, the risk of death from cold spells far exceeds that from heat waves (see the many reviews we have posted on this topic confirming this fact in our Subject Index under the heading Health Effects of Temperature: Hot vs, Cold Weather). As such, therefore, a little global warming would likely result in a net saving of lives by reducing the number of deaths that occur at the cold end of the temperature spectrum.

Antonio Gasparinni (2015) was lead author for a large international group of researchers who studied the effect of temperature extremes on death rates. Gasparrini and his co-authors analyzed data from 384 locations including the countries of Australia, Brazil, Canada, China, Italy, Japan, South Korea, Spain, Sweden, Taiwan, Thailand, the United Kingdom and the United States of America. By fitting a standard time-series Poisson model to the data obtained for each location, while controlling for trends and day of the week, they estimated temperature-mortality associations with a distributed lag non-linear model with 21 days of lag, after which they pooled the results they obtained in a multivariate meta-regression that included country indicators and temperature averages and ranges.

This work allowed them to calculate the number of human deaths attributable to heat and cold -- defined as temperatures above and below the optimum (minimum mortality) temperature -- for both moderate and extreme temperatures, the latter being defined "using cutoffs at the 2.5th and 97.5th temperature percentiles." And what did they thereby learn?

Based on data pertaining to a total of 74,225,200 human deaths that occurred between 1985 and 2012, the 23 researchers determined that 7.71% of the lives lost were caused by non-optimum temperatures; and among this group they found that "more temperature-attributable deaths were caused by cold (7.29%) than by heat (0.42%)" which makes cold in excess of seventeen times more deadly than heat. And they add, in this regard, that moderate "hot and cold temperatures represented most of the total health burden." Consequently, it seems pretty clear that any successful attempt to reverse or slow any potential increase in Earth's mean global temperature would likely come at a net cost of many human lives the world over, not a savings. The Gasparrini research provides a compelling confirmation of the reality that warmer temperatures are better for human welfare than cooler or colder temperatures. (Gasparrini Lancet 2015)

Keating and Donaldson (2001) explain that "cold causes mortality mainly from arterial thrombosis and respiratory disease, attributable in turn to cold-induced hemoconcentration and hypertension [in the first case] and respiratory infections [in the second case]." McGregor (2005) notes "anomalous cold stress can increase blood viscosity and blood pressure due to the activation of the sympathetic nervous system which accelerates the heart rate and increases vascular resistance (Collins et al., 1985; Jehn et al., 2002; Healy, 2003; Keatinge et al., 1984; Mercer, 2003; Woodhouse et al., 1993)," adding, "anomalously cold winters may also increase other risk factors for heart disease such as blood clotting or fibrinogen concentration, red blood cell count per volume and plasma cholesterol."

Wang et al. (2013) write, "A large change in temperature within one day may cause a sudden change in the heart rate and circulation of elderly people, which all may act to increase the risk of cardiopulmonary and other diseases, even leading to fatal consequences." This is significant for the climate change debate because, as Wang et al. also observe, "it has been shown that a rise of the minimum temperature has occurred at a rate three times that of the maximum temperature during the twentieth century over most parts of the world, which has led to a decrease of the diurnal temperature range (Karl et al., 1984, 1991)."

Robeson (2002) demonstrated, based on a 50-year study of daily temperatures at more than 1,000 U.S.

weather stations that daily (diurnal) temperature variability declines with warming and at a very substantial rate, so this aspect of a warmer world would lead to a reduction in temperature- related deaths.
Clearly, cold spells kill; and as has been found in almost every study of the subject, the risk of death from cold spells far exceeds that from heat waves. As such, therefore, a little global warming would likely result in a net saving of lives by reducing the number of deaths that occur at the cold end of the temperature spectrum.

Keatinge and Donaldson (2004) report coronary and cerebral thrombosis account for about half of all cold-related deaths, and respiratory diseases account for approximately half of the rest. They say cold stress causes an increase in arterial thrombosis "because the blood becomes more concentrated, and so more liable to clot during exposure to cold." As they describe it, "the body's first adjustment to cold stress is to shut down blood flow to the skin to conserve body heat," which "produces an excess of blood in central parts of the body," and to correct for this effect, "salt and water are moved out from the blood into tissue spaces," leaving behind "increased levels of red cells, white cells, platelets and fibrinogen" that lead to increased viscosity of the blood and a greater risk of clotting.
Keatinge and Donaldson also note "cold spells are closely associated with sharp increases in mortality rates," and "deaths continue for many days after a cold spell ends." On the other hand, they report, "increased deaths during a few days of hot weather are followed by a lower than normal mortality rate," because "many of those dying in the heat are already seriously ill
and even without heat stress would have died within the next 2 or 3 weeks."
With respect to the implications of global warming for human mortality, Keatinge and Donaldson state "since heat-related deaths are generally much fewer than cold-related deaths, the overall effect of global warming on health can be expected to be a beneficial one." They report, "The rise in temperature of 3.6°F expected over the next 50 years would increase heat-related deaths in Britain by about 2,000 but reduce cold-related deaths by about 20,000."
Keating and Donaldson's reference to deaths that typically would have occurred shortly even without excess heat is a phenomenon researchers call "displacement" or "harvesting." A study from Germany found "cold spells lead to excess mortality to a relatively small degree, which lasts for weeks," while "the mortality increase during heat waves is more pronounced, but is followed by lower than average values in subsequent weeks" (Laschewski and Jendritzky, 2002). The authors say the latter observation suggests people who died from short-term exposure to heat possibly "would have died in the short term anyway." They found the mean duration of above-normal mortality for the 51 heat episodes that occurred from 1968 to 1997 was 10 days, with a mean increase in mortality of 3.9%, after which there was a mean decrease in mortality of 2.3% for 19 days. Hence, the net effect of the two perturbations was an overall decrease in mortality of 0.2% over the full 29-day period.
The US EPA web site discussion of heat wave deaths referenced below reveals that the EPA recognizes heat wave deaths are not reliably counted because of loose death certificate definitions of heat caused versus heat related. Cardiovascular deaths is used as a catch all descriptor Although the deaths attributed to severe heat waves are described as Cardiovascular, the mechanism is metabolic and physiologic dysfunction and a collapse of the systems that maintain temperature equilibrium in endotherms like humans. The victims don't die of a heart attack, a coronary ischemic event caused by clots and narrowed coronary arteries, an occlusive event, they die of temperature effects and the failure of internal systems, including lung and cardiovascular system, solid organ, and brain malfunctions in the face of heat stress, dehydration, and rising core temperatures, along with dehydration and loss of mechanisms to maintain normal temperature. The victims are debilitated, and live in a stressfully hot environment and succumb for failure to acclimate and maintain normal body physiology.

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Observational Research in Asia

Behar (2000) studied sudden cardiac death (SCD) and acute myocardial infarction (AMI) in Israel, concentrating on the role temperature may play in the incidence of these health problems. Behar notes "most of the recent papers on this topic have concluded that a peak of SCD, AMI and other cardiovascular conditions is usually observed in low temperature weather during winter." He cites an Israeli study by Green et al. (1994), which reported between 1976 and 1985 "mortality from cardio-vascular disease was higher by 50% in mid-winter than in mid-summer, both in men and women and in different age groups," even though summer temperatures in the Negev, where much of the work was conducted, often exceed 30°C and winter temperatures typically do not drop below 10°C. Behar concludes these results "are reassuring for populations living in hot countries."

Kan et al. (2003) investigated the association between temperature and daily in Shanghai, China, finding a V-like relationship between total mortality and temperature that had a minimum mortality risk at 26.7°C. Above this optimum temperature, they observe, "total mortality increased by 0.73% for each degree Celsius increase; while for temperatures below the optimum value, total mortality decreased by 1.21% for each degree Celsius increase." The net effect of a warming in Shanghai, China, therefore, would likely be reduced mortality on the order of 0.5% per degree Celsius increase in temperature, or perhaps more.

Guo et al. (2012) examine the nonlinear and delayed effects of temperature on cause- specific and age-specific mortality employing data from 1999 to 2008 for Chiang Mai, Thailand with a population of 1.6 million people. Controlling for season, humidity, ozone, and particulate matter (PM10) pollution, the three researchers found "both hot and cold temperatures resulted in immediate increase in all mortality types and age groups," but "the hot effects on all mortality types and age groups were short-term, while the cold effects lasted longer." The cold effects were greater, with more people dying from them than from the effects of heat.

Lindeboom et al. (2012) used daily mortality and weather data for the period 1983–2009 pertaining to Matlab, Bangladesh, to measure lagged effects of weather on mortality, controlling for time trends and

seasonal patterns. The four researchers report "mortality in the Matlab surveillance area shows overall weak associations with rainfall, and stronger negative association with temperature." They determined there was "a 1.4% increase in mortality with every 1°C decrease in mean temperature at temperatures below 29.2°C," but only "a 0.2% increase in mortality with every 1°C increase in mean temperature."

Wang et al. (2013) evaluated the short-term effect of diurnal temperature range (DTR) on emergency room (ER) admissions among elderly adults in Beijing. The nine researchers report "significant associations were found between DTR and four major causes of daily ER admissions among elderly adults in Beijing." They state "a 1°C increase in the 8-day moving average of DTR (lag 07) corresponded to an increase of 2.08% in respiratory ER admissions and 2.14% in digestive ER admissions," and "a 1°C increase in the 3-day and 6-day moving average of DTR (lag 02 and lag 05) corresponded to a 0.76% increase in cardiovascular ER admissions, and a 1.81% increase in genitourinary ER admissions, respectively.

Wu et al. (2013) assessed the health effects of temperature on mortality in four subtropical cities of China (Changsha, Kunming, Guangzhou, and Zhuhai). The 11 researchers report a U-shaped relationship between temperature and mortality was found in the four cities, indicating "mortality is usually lowest around a certain temperature and higher at lower or higher temperatures." Although "both low and high temperatures were associated with increased mortality in the four subtropical Chinese cities," Wu et al. state the "cold effect was more durable and pronounced than the hot effect."

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Observational Research in Europe

Keatinge and Donaldson (2001) analyzed the effects on human mortality of temperature, wind, rain, humidity, and sunshine during high pollution days in the greater London area over the period 1976–1995. They observed simple plots of mortality rate versus daily air temperature revealed a linear increase as temperatures fell from 15°C to near 0°C. Mortality rates at temperatures above 15°C, however, were

"grossly alinear," as they describe it, showing no trend. Only low temperatures were found to have a significant effect on immediate and long-term mortality. They conclude "the large, delayed increase in mortality after low temperature is specifically associated with cold and is not due to associated patterns of wind, rain, humidity, sunshine, SO2, CO, or smoke."

Kysely and Huth (2004) calculated deviations of the observed number of deaths from the expected number of deaths for each day of the year in the Czech Republic for the period 1992– 2000. They found "the distribution of days with the highest excess mortality in a year is clearly bimodal, showing a main peak in late winter and a secondary one in summer." Regarding the smaller number of summer heat-wave-induced deaths, they also found "a large portion of the mortality increase is associated with the harvesting effect, which consists in short-term shifts in mortality and leads to a decline in the number of deaths after hot periods (e.g. Rooney et al., 1998; Braga et al., 2002; Laschewski and Jendritzky, 2002)." For the Czech Republic, they report, "the mortality displacement effect in the severe 1994 heat waves can be estimated to account for about 50% of the total number of victims." As they describe it, "people who would have died in the short term even in the absence of oppressive weather conditions made up about half of the total number of deaths."

Diaz et al. (2005) examined the effect of extreme winter temperature on mortality in Madrid, Spain for people older than 65, using data from 1,815 winter days over the period 1986– 1997, during which time 133,000 deaths occurred. They found that as maximum daily temperature dropped below 6°C, which they describe as an unusually cold day (UCD), "the impact on mortality also increased significantly." They also found the impact of UCDs increased as the winter progressed, with the first UCD of the season producing an average of 102 deaths/day at a lag of eight days and the sixth UCD producing an average of 123 deaths/day at a lag of eight days.

Laaidi et al. (2006) conducted an observational population study in six regions of France between 1991 and 1995 to assess the relationship between temperature and mortality in areas of widely varying climatic conditions and lifestyles. In all cases they found "more evidence was collected showing that cold weather was more deadly than hot weather." These findings, the researchers say, are "broadly consistent with those found in earlier studies conducted elsewhere in Europe (Kunst et al., 1993; Ballester et al., 1997; Eurowinter Group, 1997; Keatinge et al., 2000; Beniston, 2002; Muggeo and Vigotti, 2002), the United States (Curriero et al., 2002) and South America (Gouveia et al., 2003)." They also say their findings "give grounds for confidence in the near future," stating even a 2°C warming over the next half century "would not increase annual mortality rates."

Analitis et al. (2008) analyzed short-term effects of cold weather on mortality in 15 major European cities using data from 1990–2000, and found "a 1°C decrease in temperature was associated with a 1.35% increase in the daily number of total natural deaths and a 1.72%, 3.30% and 1.25% increase in cardiovascular, respiratory, and cerebro-vascular deaths, respectively." In addition, they report "the increase was greater for the older age groups," and the cold effect "persisted up to 23 days, with no evidence of mortality displacement." They conclude their results "add evidence that cold-related mortality is an important public health problem across Europe and should not be overlooked by public health authorities because of the recent focus on heat-wave episodes."

Wichmann et al. (2011) investigated the association between the daily three-hour maximum apparent temperature (which reflects the physiological experience of combined exposure to humidity and temperature) and deaths due to cardiovascular disease (CVD), cerebrovascular disease (CBD), and respiratory disease (RD) in Copenhagen over the period
1999–2006.

Monthly deaths in the Castile-Leon region of Spain attributable to cardiovascular disease.

Source: Adapted from Fernandez-Raga et al. (2010).

During the warm half of the year (April–September), they found a rise in temperature had an inverse or protective effect with respect to CVD mortality (a 1% decrease in death in response to a 1°C increase in apparent temperature). This finding is unusual but also has been observed in Dublin, Ireland, as reported by Baccini et al. (2008, 2011). Wichmann et al. found no association with RD and CBD mortality. At the other end of the thermal spectrum, during the cold half of the year, all three associations were inverse or protective. This finding, according to the researchers, is "consistent with other studies (Eurowinter Group, 1997; Nafstad et al., 2001; Braga et al., 2002; O'Neill et al., 2003; Analitis et al., 2008)."
Matzarakis et al. (2011) studied the relationship between heat stress and all-cause mortality in the densely populated city of Vienna (Austria). Based on data from 1970–2007, and after adjusting the long-term mortality rate to account for temporal variations in the size of the population of Vienna, temporal changes in life expectancy, and the changing age structure of Vienna's population, the three researchers found a significant relationship between heat stress and mortality. However, over this 38-year period, "some significant decreases of the sensitivity were found, especially in the medium heat stress levels," they report. These decreases in sensitivity, they write, "could indicate active processes of long-term adaptation to the increasing heat stress." In the discussion section of their paper, they write such sensitivity changes "were also found for other regions," citing Davis et al. (2003), Koppe (2005), Tan et al. (2007), and Donaldson and Keatinge (2008). In the conclusion of their paper, they refer to these changes as

"positive developments."
Kysely and Plavcova then examined "temporal changes in mortality associated with spells of large positive temperature anomalies (hot spells) in extended summer season in the population of the Czech Republic (Central Europe) during 1986–2009." They found declining mortality trends in spite of rising temperature trends, just the opposite of what IPCC claims will occur in response to global warming. The Czech scientists add, "the finding on reduced vulnerability of the population remains unchanged if possible confounding effects of within- season acclimatization and mortality displacement are taken into account," and "neither does it depend on the changing age structure of the population, since similar (and slightly more pronounced) declines in the mortality impacts are found in the elderly (age group 70+ years) when examined separately."

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Observational Research in North America

Goklany and Straja (2000) examined trends in United States death rates over the period 1979– 1997 due to excessive hot and cold weather. They report there were no trends in deaths due to either extreme heat or cold in the entire population or in the older, more-susceptible age groups, those aged 65 and over, 75 and over, and 85 and over. Deaths due to extreme cold in these older age groups exceeded those due to extreme heat by as much as 80% to 125%. With respect to the absence of trends in death rates attributable to either extreme heat or cold, Goklany and Straja say this "suggests that adaptation and technological change may be just as important determinants of such trends as more obvious meteorological and demographic factors."
Davis et al. (2003) evaluated "annual excess mortality on days when apparent temperatures—an index that combines air temperature and humidity—exceeded a threshold value for 28 major metropolitan areas in the United States from 1964 through 1998." They found "for the 28-city average, there were 41.0 ± 4.8 excess heat-related deaths per year (per standard million) in the 1960s and 1970s, 17.3 ± 2.7 in the 1980s, and 10.5 ± 2.0 in the 1990s," a remarkable decline. They conclude, "heat-related mortality in the United States seems to be largely preventable at present."
Davis et al. (2004) examined the seasonality of mortality due to all causes, using monthly data for 28 major U.S. cities from 1964 to 1998, and then calculated the consequences of a future 1°C warming of the conglomerate of those cities. At all locations studied, they report "warmer months have significantly lower mortality rates than colder months." They calculate "a uniform 1°C warming results in a net mortality decline of 2.65 deaths (per standard million) per metropolitan statistical area" (emphasis added). The primary implication of Davis et al.'s findings, in their words, "is that the seasonal mortality pattern in US cities is largely independent of the climate and thus insensitive to climate fluctuations, including changes related to increasing greenhouse gases."
Deschenes and Moretti (2009) analyzed the relationship between weather and mortality, based on "data that include the universe of deaths in the United States over the period 1972– 1988," in which they "match each death to weather conditions on the day of death and in the county of occurrence." They discovered "hot temperature shocks are indeed associated with a large and immediate spike in mortality in the days of the heat wave," but "almost all of this excess mortality is explained by near-term displacement." As a result, "in the weeks that follow a heat wave, we find a marked decline in mortality hazard, which completely offsets the increase during the days of the heat wave," so "there is virtually no lasting impact of heat waves on mortality." In the case of cold temperature days, they also found "an immediate spike in mortality but "there is no offsetting decline in the weeks that follow," so "the cumulative effect of one day of extreme cold temperature during a thirty-day window is an increase in daily mortality by as much as 10%."

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Global Warming and Cardiovascular Disease

The key findings are that

- Global warming, if it does occur, would reduce the incidence of fatal coronary events related to low temperatures and wintry weather by a much greater degree than it increases the incidence of death or serious heat related events associated with high temperatures and summer heat waves.

- Non-fatal myocardial infarction is also less frequent during unseasonably warm periods than during unseasonably cold periods.

- Any cost-benefit analysis that attributes an increase in cardiovascular events to warming is incorrect. Heat illness injures and kills by other means and has a much lesser death toll proportionately than cold related events. Heat illness injury and death in heat waves affects the debilitated and chronically ill in hot unventilated environments and the mechanism is dehydration and loss of core body temperature control.

Cardiovascular diseases affect the heart and or the blood vessels. They include arrhythmia, arteriosclerosis, congenital heart disease, and coronary artery disease, diseases of the aorta and its branches, disorders of the peripheral vascular system, endocarditis, heart valve disease, hypertension, orthostatic hypotension, and

shock. According to IPCC, exposure to rising temperatures and especially heat waves can cause premature deaths due to heat-induced illness. The claims that it causes stroke or myocardial infarctions are not correct except to concede that ultimately most deaths are cardiovascular in nature.
Empirical research suggests that heat illness can cause collapse and death, but the mechanism is fluid and circulatory collapse, not stroke or heart attack. Heat stroke is severe heat illness with loss of temperature control that produces brain dysfunction; it's not a cerebral thrombosis or hemorrhage, a true stroke.
That aside, the IPCC overlooks the fact that cooler temperatures cause an even larger number of premature deaths, with the result that a warmer world would experience fewer deaths in total due to cardiovascular disease.

Global Warming and Respiratory Disease

The key findings of this section include the following:

- Global warming, if it did occur would reduce incidence of death due to respiratory disease around the world, for example the Americas, Spain, Canada, Shanghai, and even on the subtropical island of Taiwan.

- Lower minimum temperatures are a strong risk factor for outpatient visits for respiratory diseases. Warmer temperatures reduce rates of respiratory disease.

- Any cost-benefit analysis that attributes increases in deaths or disease and disability or loss of work/school time to warming is incorrect and not a reliable guide for public policy.

Respiratory diseases are diseases affecting the organs and tissues that make gas exchange possible in humans and other higher organisms. They range from the common cold, allergies, asthma, and bronchiolitis to life-threatening conditions including pneumonia, pulmonary embolism, and lung cancer. Acute respiratory disease is a condition in which breathing becomes difficult and oxygen levels in the blood drop lower than normal. Respiratory diseases are widespread. For example, childhood asthma affects more than 300 million people worldwide (Baena-Cagnani and Badellino, 2011). Non-fatal respiratory diseases impose enormous social costs due to days lost from work and school (Mourtzoukou and Falagas, 2007).
According to IPCC, rising atmospheric carbon dioxide concentrations due to the combustion of fossil fuels causes global warming, and this temperature increase causes increased deaths due to respiratory disease. However, examination of real-world data reveals unassailable evidence that colder temperatures cause more deaths and hospital admissions due to respiratory disease than do warmer temperatures.
Some of the studies cited earlier in this chapter on lower death rates due to warmer temperatures and cardiovascular disease also identified specific reductions in fatalities due to respiratory diseases, so their research also appears in this section. Keatinge and Donaldson (2001), for example, studied of the effects of temperature on mortality in people over 50 years of age in the greater London area over the period 1976–1995. Simple plots of mortality rate versus daily air temperature revealed a linear increase in mortality as the air temperature fell from 15°C to near 0°C. Mortality rates at temperatures above 15°C, on the other hand, showed no trend. The authors say it is because "cold causes mortality mainly from arterial thrombosis and respiratory disease, attributable in turn to cold-induced hemo-concentration and hypertension and respiratory infections" (emphasis added).
Nafstad et al. (2001) studied the association between temperature and daily mortality in citizens of Oslo, Norway over the period 1990 to 1995. The results showed the mean daily number of respiratory-related

deaths was considerably higher in winter (October–March) than in summer (April–September). Winter deaths associated with respiratory diseases were 47% more numerous than summer deaths. They conclude, "A milder climate would lead to a substantial reduction in average daily number of deaths." Read milder as warmer.

Hajat and Haines (2002) examined the relationship between cold temperatures and the number of visits by the elderly to general practitioners for asthma, lower respiratory diseases other than asthma, and upper respiratory diseases other than allergic rhinitis as obtained for registered patients aged 65 and older from several London practices between January 1992 and September 1995. They found the mean number of consultations was higher in cool-season months (October–March) than in warm-season months (April–September) for all respiratory diseases. At mean temperatures below 5°C, the relationship between respiratory disease consultations and temperature was linear, and stronger at a time lag of six to 15 days A 1°C decrease in mean temperature below 5°C was associated with a 10.5% increase in all respiratory disease consultations.

Braga et al. (2002) conducted a time-series analysis of both the acute and lagged influence of temperature and humidity on mortality rates in 12 U.S. cities, finding no clear evidence for a link between humidity and respiratory-related deaths. With respect to temperature, they found respiratory-related mortality increased in cities with more variable temperature. This phenomenon, they write, "suggests that increased temperature variability is the most relevant change in climate for the direct effects of weather on respiratory mortality."

Gouveia et al. (2003) extracted daily counts of deaths from all causes, except violent

deaths and neonatal deaths (up to one month of age), from Sao Paulo, Brazil's mortality information system for the period 1991–1994 and analyzed them for effects of temperature. For respiratory-induced deaths, death rates due to a 1°C cooling were twice as great as death rates due to a 1°C warming in adults and 2.8 times greater in the elderly.

Nakaji et al. (2004) evaluated seasonal trends in deaths due to various diseases in Japan, using nationwide vital statistics from 1970 to 1999 and concurrent mean monthly air temperature data. They found the numbers of deaths due to respiratory diseases, including pneumonia and influenza, rise to a maximum during the coldest time of the year. The team of nine scientists concludes, "To reduce the overall mortality rate and to prolong life expectancy in Japan, measures must be taken to reduce those mortality rates associated with seasonal differences."

Bartzokas et al. (2004) "examined the relationship between hospital admissions for cardio-vascular (cardiac in general including heart attacks) and/or respiratory diseases (asthma etc.) in a major hospital in Athens [Greece] and meteorological parameters for an 8-year period." Over the whole year, they found, "there was a dependence of admissions on temperature," and low temperatures were "responsible for a higher number of admissions." Specifically, "there was a decrease of cardiovascular or/and respiratory events from low to high values [of temperature], except for the highest temperature class in which a slight increase was recorded."

Kovats et al. (2004) studied patterns of temperature-related hospital admissions and deaths in Greater London during the mid-1990s. For the three-year period 1994–1996, they found respiratory-related deaths were nearly 150% greater in the depth of winter cold than at the height of summer warmth. They also found the mortality impact of the heat wave of 29 July to 3 August 1995 (which boosted daily mortality by just over 10%) was so tiny it could not be discerned among the random scatter of plots of three-year-average daily deaths from cardiovascular and respiratory problems versus day of year. Similarly, in a study of temperature effects on mortality in three English counties (Hampshire, West Midlands, and West Yorkshire), McGregor (2005) found "the occurrence of influenza ... helps elevate winter mortality above that of summer."

Carder et al. (2005) investigated the relationship between outside air temperature and deaths due to all non-accident causes in the three largest cities of Scotland (Glasgow, Edinburgh, and Aberdeen) between January 1981 and December 2001. The authors observed "an overall increase in mortality as temperature decreases," which "appears to be steeper at lower temperatures than at warmer temperatures," and "there is little evidence of an increase in mortality at the hot end of the temperature range." Specifically regarding respiratory disease, they found "for temperatures below 11°C, a 1°C drop in the daytime mean temperature on any one day was associated with an increase in respiratory mortality of 4.8% over the following month."
Donaldson (2006) studied the effect of annual mean daily air temperature on the length of the yearly respiratory syncytial virus (RSV) season, the virus which causes bronchiolitis, in England and Wales for 1981–2004. Reporting "climate change may be shortening the RSV season," Donaldson found "the seasons associated with laboratory isolation of respiratory syncytial virus (for 1981–2004) and RSV-related emergency department admissions (for 1990– 2004) ended 3.1 and 2.5 weeks earlier, respectively, per 1°C increase in annual central England temperature ($P = 0.002$ and 0.043, respectively)." Consequently, since "no relationship was observed between the start of each season and temperature," he reports, so "the RSV season has become shorter." He concludes, "These findings imply a health benefit of global warming in England and Wales associated with a reduction in the duration of the RSV season and its consequent impact on the health service."
Frei and Gassner (2008) studied hay fever prevalence in Switzerland from 1926 to 1991, finding it rose from just under 1% of the country's population to just over 14%, but from 1991 to 2000 it leveled off, fluctuating about a mean value on the order of 15%. The authors write, "several studies show that no further increase in asthma, hay fever and atopic sensitization in adolescents and adults has been observed during the 1990s and the beginning of the new century," citing Braun-Fahrlander et al. (2004) and Grize et al. (2006). They write, "Parallel to the increasing hay fever rate, the pollen amounts of birch and grass were increasing from 1969 to 1990," but "subsequently, the pollen of these plant species decreased from 1991 to 2007." They say this finding "is more or less consistent with the changes of the hay fever rate that no longer increased during this period and even showed a tendency to decrease slightly." Nearly identical findings were presented a year later (Frei, 2009). Although some have claimed rising temperatures and CO2 concentrations will lead to more pollen and more hay fever (Wayne et al., 2002), the analyses of Frei (2009) and Frei and Gassner (2008) suggest that is not true of Switzerland.
Miller et al. (2012) extracted annual prevalence data for frequent otitis media (defined as three or more ear infections per year), respiratory allergy, and non-respiratory seizures in children from the U.S. National Health Interview Survey for 1998 to 2006. They also obtained average annual temperatures for the same period from the U.S. Environmental Protection Agency. They found "annual temperature did not influence the prevalence of frequent otitis media," "annual temperature did not influence prevalence of respiratory allergy," and "annual temperature and sex did not influence seizure prevalence." They conclude their findings "may demonstrate that average temperature is not likely to be the dominant cause of the increase in allergy burden or that larger changes in temperatures over a longer period are needed to observe this association." They further conclude, "In the absence of more dramatic annual temperature changes, we do not expect prevalence of otitis media to change significantly as global warming may continue to affect our environment."
Xu et al. (2013) examined the relationship between diurnal temperature range (DTR) and emergency department admissions for childhood asthma in Brisbane, Australia, from January 1st 2003 to December 31st 2009. The six scientists report "childhood asthma increased above a DTR of 10°C" and "was the greatest for lag 0–9 days, with a 31% increase in [hospital] emergency department admissions per 5°C increment of DTR," further noting, "male children and children aged 5–9 years appeared to be more vulnerable to the DTR effect than others."

Ge et al. (2013) also investigated respiratory health and DTR. The researchers collected numbers of daily emergency-room visits for RTI at one of the largest medical establishments in Shanghai, China (Huashan Hospital) between 1 January 2008 and 30 June 2009, along with DTR data and data pertaining to possible confounding air pollutants (PM10, SO2, and NO2). After making appropriate statistical analyses, the scientists determined increasing DTRs were closely associated with daily emergency-room visits for RTIs, such that "an increase of 1°C in the current-day and in the 2-day moving average DTR corresponded to a 0.94% and 2.08% increase in emergency-room visits for RTI, respectively."
Lin et al. (2013) used data on daily area-specific deaths from all causes, circulatory diseases, and respiratory diseases in Taiwan, developing relationships between each of these cause-of-death categories and a number of cold-temperature related parameters for 2000–2008. The five researchers discovered "mortality from [1] all causes and [2] circulatory diseases and [3] outpatient visits of respiratory diseases has a strong association with cold temperatures in the subtropical island, Taiwan." In addition, they found "minimum temperature estimated the strongest risk associated with outpatient visits of respiratory diseases."

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Global Warming and Strokes

The key findings of this section include the following:

- Any warming would reduce the incidence of death due to stroke in many parts of the world, including Russia, Korea, Japan, Africa, Asia, Europe, Latin America, and the Caribbean.

- Low minimum temperatures are a stronger risk factor than high temperatures for stroke incidence and hospitalization.

- Any cost-benefit analysis that attributes increased strokes to a prediction of global warming is incorrect and not a reliable guide for public policy.

A stroke occurs when blood flow to an area in the brain is cut off. Ischemic stroke occurs when clots form in the brain's blood vessels, in blood vessels leading to the brain, or in blood vessels elsewhere in the body and then travel to the brain. Ischemic stroke can also occur when too much plaque (fatty deposits and cholesterol) clogs the brain's blood vessels. Hemorrhagic strokes occur when a blood vessel in the brain breaks or ruptures. The result is blood seeping into the brain tissue, causing damage to brain cells. The most common causes of hemorrhagic stroke are high blood pressure and brain aneurysms. An aneurysm is a bulge in a blood vessel caused by a weakness and thinning of the blood vessel wall. Aneurysms are prone to burst and a major cause of hemorrhagic stroke (WebMD, 2015).
According to IPCC, rising atmospheric carbon dioxide concentrations due to the combustion of fossil fuels causes global warming, and this temperature increase causes increased deaths due to strokes. Not true. Examination of real-world data reveals unseasonable cold temperatures cause more deaths and hospital admissions due to stroke than do unseasonable warm temperatures.
Feigin et al. (2000) examined the relationship between the incidence of stroke and ambient temperatures over the period 1982-1993 in Novosibirsk, Siberia, which has one of the highest stroke incidence rates in the world. Based on analyses of 2,208 patients with sex and age distributions similar to those of Russia as a whole, they found a statistically significant association between stroke and low ambient temperature. In the

case of ischemic stroke (IS), which accounted for 87% of all stroke types, they determined "the risk of IS occurrence on days with low ambient temperature [was] 32% higher than that on days with high ambient temperature." They conclude the "very high stroke incidence in Novosibirsk, Russia may partially be explained by the highly prevalent cold factor there." There is no reason to believe that temperature variations would have a discernible effect on hemorrhagic strokes that occur because of vascular pathology, not occlusion.

Hong et al. (2003) investigated the association between the onset of ischemic stroke and prior episodic decreases in temperature in 545 patients who suffered strokes in Incheon, Korea from January 1998 to December 2000. They report "decreased ambient temperature was associated with risk of acute ischemic stroke," with the strongest effect being seen on the day after exposure to cold weather, further noting "even a moderate decrease in temperature can increase the risk of ischemic stroke." They also found "risk estimates associated with decreased temperature were greater in winter than in the summer," which suggests "low temperatures as well as temperature changes are associated with the onset of ischemic stroke." Finally, they explain the reason for the 24- to 48-hour lag between exposure to cold and the onset of stroke "might be that it takes some time for the decreasing temperature to affect blood viscosity or coagulation.

Nakaji et al. (2004) evaluated seasonal trends in deaths due to various diseases in Japan using nationwide vital statistics from 1970 to 1999 together with mean monthly temperature data. They found the peak mortality rate due to stroke was two times greater in winter (January) than at the time of its yearly minimum (August and September).

Chang et al. (2004) analyzed data from the World Health Organization (WHO) Collaborative Study of Cardiovascular Disease and Steroid Hormone Contraception (WHO, 1995) to determine the effects of monthly mean temperature on rates of hospitalization for arterial stroke and acute myocardial infarction among women aged 15–49 from 17 countries in Africa, Asia, Europe, Latin America, and the Caribbean. They found among these women, a 5°C reduction in mean air temperature was associated with a 7% increase in the expected hospitalization rate due to stroke, and this effect was relatively acute, within a period of about a month, the scientists write.

Gill et al. (2012) write, "in the past two decades, several studies reported that meteorologic changes are associated with monthly and seasonal spikes in the incidence of aneurysmal subarachnoid hemorrhage (aSAH)," and "analysis of data from large regional databases in both hemispheres has revealed increased seasonal risk for aSAH in the fall, winter and spring," citing among other sources Feigin et al. (2001), Abe et al. (2008), and Beseoglu et al. (2008). Gill et al. identified the medical records of 1,175 patients at the Johns Hopkins Hospital in Baltimore, Maryland (USA) who were admitted with a radiologically confirmed diagnosis of aSAH between 1 January 1991 and 1 March 2009. The six scientists report both "a one-day decrease in temperature and colder daily temperatures were associated with an increased risk of incident aSAH," and "these variables appeared to act synergistically" and were "particularly predominant in the fall, when the transition from warmer to colder temperatures occurred." Gill et al. add their study "is the first to report a direct relationship between a temperature decrease and an increased risk of aSAH," and "it also confirms the observations of several reports of an increased risk of aSAH in cold weather or winter," citing Nyquist et al. (2001) and other sources. Authors' note: This study and others the authors of the study reference are outliers in the sense that they tally aneurysmal sub arachnoid hemorrhage, a different kind of stroke than ischemic strokes, so there is no "mechanism" of coagulation and clot formation that would relate to temperature that might be hypothesized as a cause of cold or cool to cause hemorrhagic stroke.

The reader should be informed that hemorrhagic stroke is because of a different mechanism, the rupture of a weakened wall of a blood vessel, often associated with a bulge called an aneurysm, as opposed to ischemic stroke discussed above that occur because of a blood clot in the brain blood vessel. However the temperature

effect is the same, cold produces an increase in hemorrhagic strokes in addition to its effect on the rate of ischemic strokes.

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Global Warming and Insect-borne Diseases

The key findings of this section include the following:

- Research contradicts the claim that malaria will expand across the globe and intensify as a result of any possible warming.

- Concerns over large increases in dengue fever as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue fever infection rates.

- Climate change has not been a significant factor driving the recent temporal patterns in the epidemiology of tick-borne diseases. Ticks are endemic at many latitudes.

The latest IPCC report, the Fifth Assessment Report (AR5) backs down from previous predictions that global warming would facilitate the spread of insect-borne diseases including malaria, dengue fever, and tick-borne diseases. The full report from Working Group II on the subject (IPCC, 2014a, Chapter 11, pp. 722-726) repeatedly admits there is no evidence that climate change has affected the range of vector-borne diseases including tick-borne diseases. However, the Summary for Policymakers inexplicably warns "Throughout the 21st century, climate change is expected to lead to increases in ill-health in many regions and especially in developing countries with low income, as compared to a baseline without climate change (high confidence)." Among the "examples" given is "vector-borne diseases (medium confidence)" (IPCC, 2014b, pp. 19-20). Such predictions are not supported by the evidence.

In a research report in Science, Rogers and Randolph (2000) note "predictions of global climate change have stimulated forecasts that vector-borne diseases will spread into regions that are at present too cool for their persistence." However, the effect of warmer temperatures on insect-borne diseases is complex, sometimes working in favor of and sometimes against the spread of a disease. For example, ambient temperature has historically not determined the range of insect-borne diseases, hotter weather shortens the lifespan of mosquitos, and human adaptation as well as vector control measures can neutralize any detrimental effect of warming, to overwhelm the role of climate. Even those who support IPCC, such as Marm Kilpatrick, an assistant professor in ecology and evolutionary biology at the University of California, Santa Cruz, admits "It's a little bit tricky to make a solid prediction" (Irfan, 2011).

Gething et al. (2010), writing specifically about malaria, may have put it best when they said there has been "a decoupling of the geographical climate-malaria relationship over the twentieth century, indicating that non-climatic factors have profoundly confounded this relationship over time." They note "non-climatic factors, primarily direct disease control and the indirect effects of a century of urbanization and economic development, although spatially and temporally variable, have exerted a substantially greater influence on the geographic extent and intensity of malaria worldwide during the twentieth century than have climatic factors." As for the future, they conclude climate-induced effects "can be offset by moderate increases in coverage levels of currently available interventions."

This section investigates the reliability of IPCC's claim with respect to the three main kinds of insect-borne diseases: malaria, dengue fever, and tick-borne diseases According to the results of a vast body of scientific examination and research on this topic, there is little support for the claims appearing in the latest IPCC Summary for Policymakers.

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Malaria

A vast body of scientific examination and research contradict the claim that malaria will expand across the globe and intensify as a result of CO2-induced warming.

Jackson et al. (2010) say "malaria is one of the most devastating vector-borne parasitic diseases in the tropical and subtropical regions of the world," noting it affects more than 100 countries.

According to the World Health Organization, Africa carries the highest infection burden of any continent, with nearly 200 million cases reported in 2006, and the Centers for Disease Control and Prevention estimates between 700,000 and 2.7 million people each year die from the dreaded disease (Suh et al., 2004). In addition, Jackson et al. report "the African region bears 90% of these estimated worldwide deaths," and "three-quarters of all malaria related deaths are among African children," citing Breman (2001). According to Reiter (2000), claims that malaria resurgence is the product of CO2-induced global warming ignore other important factors and disregard known facts. A historical analysis of malaria trends, for example, reveals this disease was an important cause of illness and death in England during a period of colder-than-present temperatures throughout the Little Ice Age. Its transmission began to decline only in the nineteenth century, during a warming phase, when, according to Reiter, "temperatures were already much higher than in the Little Ice Age." In short, malaria was prevalent in Europe during some of the coldest centuries of the past millennium, and it has only recently undergone widespread decline, when temperatures have been warming.

Clearly, there are other factors at work in regards to malaria that are more important than temperature. Such factors include the quality of public health services, irrigation and agricultural activities, land use practices, civil strife, natural disasters, ecological change, population change, use of insecticides, and the movement of people (Reiter, 2000; Reiter, 2001; Hay et al., 2002).

Nevertheless, concerns have lingered about the possibility of widespread future increases in malaria due to global warming. These concerns are generally rooted in climate models that typically use only one, or at most two, climate variables in making their predictions of the future distribution of the disease over Earth, and they generally do not include any of the non-climatic factors listed in the preceding paragraph. When more variables are included, a less-worrisome future is projected.

In one modeling study, for example, Rogers and Randolph (2000) employed five climate variables and obtained very different results. Briefly, they used the present-day distribution of malaria to determine the specific climatic constraints that best define that distribution, after which the multivariate relationship they derived from this exercise was applied to future climate scenarios derived from state-of-the-art climate models, in order to map potential future geographical distributions of the disease.

Their study revealed very little change: a 0.84% increase in potential malaria exposure under the "medium-high" scenario of global warming and a 0.92% decrease under the "high" scenario. Rogers and Randolph explicitly state their quantitative model "contradicts prevailing forecasts of global malaria expansion" and "highlights the use of multivariate rather than univariate constraints in such applications. They found "climate warming, expressed as a systematic temperature increase over the 85-year period, does not appear to be responsible for an increase in malaria suitability over any region in Africa." They conclude "research on the links between climate change and the recent resurgence of malaria across Africa would be best served through refinements in maps and models of precipitation patterns and through closer examination of the role of nonclimatic influences."

Kuhn et al. (2003) analyzed the determinants of temporal trends in malaria deaths within England and Wales in 1840–1910 and found "a 1°C increase or decrease was responsible for an increase in malaria deaths of 8.3% or a decrease of 6.5%, respectively," which explains "the malaria epidemics in the 'unusually hot summers' of 1848 and 1859." Nevertheless, the long- term near-linear temporal decline in malaria deaths over the period of study, the researchers write, "was probably driven by nonclimatic factors," among which they identify increasing livestock populations (which tend to divert mosquito biting from humans), decreasing acreages of marsh wetlands (where mosquitoes breed), as well as "improved housing, better access to health care and medication, and improved nutrition, sanitation, and hygiene." Kuhn et al. say "the projected increase in proportional risk is clearly insufficient to lead to the reestablishment of endemicity."

Childs et al. (2006) present a detailed analysis of malaria incidence in northern Thailand based on a quarter-century monthly time series (January 1977 through January 2002) of total malaria cases in the country's 13 Northern provinces. Over this time period, when IPCC claims the world warmed at a rate and to a level unprecedented over the prior one to two millennia, Childs et al. report there was an approximately constant rate of decline in total malaria incidence (from a mean monthly incidence in 1977 of 41.5 cases per hundred thousand people to 6.72 cases per hundred thousand people in 2001). Noting "there has been a steady reduction through time of total malaria incidence in northern Thailand, with an average decline of 6.45% per year," they say this result "reflects changing agronomic practices and patterns of immigration, as well as the success of interventions such as vector control programs, improved availability of treatment and changing drug policies."

Reiter (2008) came to similar conclusions, writing "simplistic reasoning on the future prevalence of malaria is ill-founded; malaria is not limited by climate in most temperate regions, nor in the tropics, and in nearly all cases, 'new' malaria at high altitudes is well below the maximum altitudinal limits for transmission." He further states, "Future changes in climate may alter the prevalence and incidence of the disease, but

obsessive emphasis on 'global warming' as a dominant parameter is indefensible; the principal determinants are linked to ecological and societal change, politics and economics."
Hulden and Hulden (2009) analyzed malaria statistics collected in Finland from 1750 to 2008 via correlation analyses between malaria frequency per million people and all variables that have been used in similar studies throughout other parts of Europe, including temperature data, animal husbandry, consolidation of land by redistribution, and household size. Over the entire period, "malaria frequency decreased from about 20,000–50,000 per 1,000,000 people to less than 1 per 1,000,000 people," they report. The two Finnish researchers conclude, "Indigenous malaria in Finland faded out evenly in the whole country during 200 years with limited or no counter measures or medication," making that situation "one of the very few opportunities where natural malaria dynamics can be studied in detail." Their study indicates "malaria in Finland basically was a sociological disease and that malaria trends were strongly linked to changes in the human household size and housing standard."

Effects of climate and socioeconomic factors on the projected future global distribution of malaria.

Source: Béguin et al. (2011).

The many findings described above make it clear a vast body of scientific examination and research contradict the claim that malaria will expand across the globe and intensify as a result of CO2-induced warming.

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Dengue Fever

Concerns over large increases in dengue fever as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue fever.

According to Ooi and Gubler (2009), "dengue/dengue hemorrhagic fever is the most important vector-borne viral disease globally," with more than half the world's population living in areas deemed to be at risk of infection. Kyle and Harris (2008) note "dengue is a spectrum of disease caused by four serotypes of the most

prevalent arthropod-borne virus affecting humans today," and "its incidence has increased dramatically in the past 50 years," to where "tens of millions of cases of dengue fever are estimated to occur annually, including up to 500,000 cases of the life- threatening dengue hemorrhagic fever/dengue shock syndrome." Some of the research papers summarized in previous sections address dengue fever as well as malaria. With a few worthy exceptions, we do not repeat those summaries in this section. The most important exceptions are papers written by or coauthored by Paul Reiter (2001, 2003, 2010a, 2010b), one of the world's premier authorities on the subject. Reiter analyzed the history of malaria and dengue fever in an attempt to determine whether the incidence and range of influence of these diseases would indeed increase in response to CO2-induced global warming.

His reviews established what is now widely accepted among experts in the field, that the natural history of these vector-borne diseases is highly complex, and the interplay of climate, ecology, vector biology, and a number of other factors defy definition by the simplistic analyses utilized in the computer models relied on by environmental activists and the IPCC.

That there has in fact been a resurgence of these diseases in parts of the world is true, but as Reiter (2001) notes; it is "facile to attribute this resurgence to climate change." This he shows via a number of independent analyses that clearly demonstrate factors associated with politics, economics, and human activity is the principal determinants of the spread of these diseases. He describes these factors as being "much more significant" than climate in promoting disease expansion. Two years later, Reiter took up the subject again, this time with 19 other scientists as coauthors (Reiter et al., 2003), and yet again in 2010. Reiter's work remains the most comprehensive critique of the claims of the Intergovernmental Panel on Climate Change.

Kyle and Harris (2008) wrote "there has been a great deal of debate on the implications of global warming for human health," but "at the moment, there is no consensus." However, "in the case of dengue," they report, "it is important to note that even if global warming does not cause the mosquito vectors to expand their geographic range, there could still be a significant impact on transmission in endemic regions," because "a 2°C increase in temperature would simultaneously lengthen the lifespan of the mosquito and shorten the extrinsic incubation period of the dengue virus, resulting in more infected mosquitoes for a longer period of time." Nevertheless, they state there are "infrastructure and socioeconomic differences that exist today and already prevent the transmission of vector-borne diseases, including dengue, even in the continued presence of their vectors," citing Reiter (2001).

Wilder-Smith and Gubler (2008) conducted a review of the scientific literature, noting "the past two decades saw an unprecedented geographic expansion of dengue" and "global climate change is commonly blamed for the resurgence of dengue," but they add, "There are no good scientific data to support this conclusion." The two researchers report, "Climate has rarely been the principal determinant of [their] prevalence or range," and "human activities and their impact on local ecology have generally been much more significant." They cite as contributing factors "urbanization, deforestation, new dams and irrigation systems, poor housing, sewage and waste management systems, and lack of reliable water systems that make it necessary to collect and store water," further noting "disruption of vector control programs, be it for reasons of political and social unrest or scientific reservations about the safety of DDT, has contributed to the resurgence of dengue around the world."

In addition, Wilder-Smith and Guble write "large populations in which viruses circulate may also allow more co-infection of mosquitoes and humans with more than one serotype of virus," which would appear to be borne out by the fact that "the number of dengue lineages has been increasing roughly in parallel with the size of the human population over the last two centuries." Most important, perhaps, is "the impact of international travel," of which they say "humans, whether troops, migrant workers, tourists, business travelers, refugees, or others, carry the virus into new geographic areas," and these movements "can lead to

epidemic waves." The two researchers conclude, "Population dynamics and viral evolution offer the most parsimonious explanation for the observed epidemic cycles of the disease, far more than climatic factors." Russell et al. (2009) showed the dengue vector (the Aedes Aegypti mosquito) "was previously common in parts of Queensland, the Northern Territory, Western Australia and New South Wales," and it had, "in the past, covered most of the climatic range theoretically available to it," adding "the distribution of local dengue transmission has [historically] nearly matched the geographic limits of the vector." This being the case, they conclude the vector's current absence from much of Australia "is not because of a lack of a favorable climate." Thus, they reason "a temperature rise of a few degrees is not alone likely to be responsible for substantial increases in the southern distribution of A. Aegypti or dengue, as has been recently proposed." Instead of futile attempts to limit dengue transmission by controlling the world's climate, therefore, the medical researchers recommend "well resourced and functioning surveillance programs, and effective public health intervention capabilities, are essential to counter threats from dengue and other mosquito-borne diseases."

Reiter (2010a) observed "the introduction and rapidly expanding range of Aedes Albopictus in Europe is an iconic example of the growing risk of the globalization of vectors and vector-borne diseases," and "the history of yellow fever and dengue in temperate regions confirms that transmission of both diseases could recur, particularly if Aedes Aegypti, a more effective vector, were to be re-introduced." He states "conditions are already suitable for transmission." Much more important than a rise or fall of a couple degrees of temperature, Reiter says, is "the quantum leap in the mobility of vectors and pathogens that has taken place in the past four decades, a direct result of the revolution of transport technologies and global travel."

Carbajo et al. (2012) evaluated the relative contributions of geographic, demographic, and climatic variables to the recent spread of dengue in Argentina. They found dengue spatial occurrence "was positively associated with days of possible transmission, human population number, population fall and distance to water bodies." When considered separately, the researchers write, "the classification performance of demographic variables was higher than that of climatic and geographic variables." Thus, although useful in estimating annual transmission risk, Carbajo et al. conclude temperature "does not fully describe the distribution of dengue occurrence at the country scale," and "when taken separately, climatic variables performed worse than geographic or demographic variables."

These several observations indicate concerns over large increases in dengue fever as a result of rising temperatures are unfounded and unsupported by the scientific literature, as climatic indices are poor predictors for dengue fever.

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Tick-borne Diseases

Climate change has not been the most significant factor driving the recent temporal patterns in the epidemiology of tick-borne diseases.

Sarah Randolph of the University of Oxford's Department of Zoology is a leading scholar on tick-borne diseases. She and fellow Oxford faculty member David Rogers observed in 2000 that tick-borne encephalitis (TBE) "is the most significant vector-borne disease in Europe and Eurasia," having "a case morbidity rate of 10–30% and a case mortality rate of typically 1–2% but as high as 24% in the Far East." The disease is

caused by a flavivirus (TBEV), which is maintained in natural rodent-tick cycles; humans may be infected with it if bitten by an infected tick or by drinking untreated milk from infected sheep or goats.

Early discussions on the relationship of TBE to global warming predicted the disease would expand its range and become more of a threat to humans in a warmer world. However, Randolph and Rogers (2000) note, "like many vector-borne pathogen cycles that depend on the interaction of so many biotic agents with each other and with their abiotic environment, enzootic cycles of TBEV have an inherent fragility," so "their continuing survival or expansion cannot be predicted from simple univariate correlations."

Randolph (2010) examined the roles played by various factors that may influence the spread of tick-borne diseases. After describing some of the outbreaks of tick-borne disease in Europe over the past couple of decades, Randolph states "the inescapable conclusion is that the observed climate change alone cannot explain the full heterogeneity in the epidemiological change, either within the Baltic States or amongst Central and Eastern European countries," citing Sumilo et al. (2007). Instead, she writes, "a nexus of interrelated causal factors—abiotic, biotic and human—has been identified," and "each factor appears to operate synergistically, but with differential force in space and time, which would inevitably generate the observed epidemiological heterogeneity."

Many of these factors, she continues, "were the unintended consequences of the fall of Soviet rule and the subsequent socio-economic transition (Sumilo et al., 2008b)," among which she cites "agricultural reforms resulting in changed land cover and land use, and an increased reliance on subsistence farming; reduction in the use of pesticides, and also in the emission of atmospheric pollution as industries collapsed; increased unemployment and poverty, but also wealth and leisure time in other sectors of the population as market forces took hold."

Randolph concludes "there is increasing evidence from detailed analyses that rapid changes in the incidence of tick-borne diseases are driven as much, if not more, by human behavior that determines exposure to infected ticks than by tick population biology that determines the abundance of infected ticks," as per Sumilo et al. (2008a) and Randolph et al. (2008). She ends her analysis by stating, "While nobody would deny the sensitivity of ticks and tick-borne disease systems to climatic factors that largely determine their geographical distributions, the evidence is that climate change has not been the most significant factor driving
the recent temporal patterns in the epidemiology of tick-borne diseases."

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Conclusion

IPCC fails to acknowledge the human health benefits of a warming world, claiming instead that the net effect of warming is a cost rather than a benefit.

Fossil fuels have benefited human health by making possible the dramatic increase in human prosperity since the first Industrial Revolution, making investments possible in goods and services that are essential to protecting human health and prolonging human life. Fossil fuels further improve human health by making environmental protection both valued and financially possible and by powering technologies and production of goods and services, transportation, communication that all improve quality of life, and protect human health and welfare, extend life spans.
If the combustion of fossil fuels leads to some amount of global warming, then the positive as well as negative health effects of that warming should be included in any cost-benefit analysis of fossil fuels.
Medical science explains why colder temperatures often cause diseases and sometimes fatalities whereas warmer temperatures are associated with health benefits.
Empirical research confirms that warmer temperatures lead to a net decrease in temperature- related mortality in virtually all parts of the world, even those with tropical climates. The evidence of this benefit comes from research conducted in nearly every major country of the world.
Global warming is reducing the incidence of fatal coronary events related to low temperatures and wintry weather by a much greater degree than it increases the incidence of heat related illness or death attributable to heat waves. Respiratory illness, strokes and myocardial infarction are less frequent during unseasonably warm periods than during unseasonably cold periods.
Global warming is reducing the incidence of death due to respiratory disease in many parts of the world, including Spain, Canada, Shanghai, and even on the subtropical island of Taiwan. Low minimum temperatures have been found to be a stronger risk factor than high temperatures for outpatient visits for respiratory diseases. Warm weather reduces the incidence of death due to stroke around the world.
A vast body of scientific examination and research contradicts and refutes the claim that malaria will expand across the globe or intensify in some regions as a result of any predicted CO2-induced warming. Concerns over large increases in mosquito-transmitted dengue fever as a result of rising temperatures are unfounded and unsupported by the scientific literature.

While climatic factors largely determine the geographical distribution of ticks, temperature and climate change are not among the significant factors determining the incidence of tick-borne diseases.
In the face of this extensive evidence of the positive effects of fossil fuels on human health, IPCC continues to claim the net impact on human health of fossil fuels will be negative. Because virtually all cost-benefit analyses incorporate the IPCC's incorrect assumptions into their calculation of the social cost of fossil fuels, they are unreliable guides to policymakers.

12. Conclusion

This is not a complete expose of the misconduct of the US EPA sponsored researchers and in house science and policy staff in matters of epidemiology and toxicology and it focuses on the US EPA research/ policy /regulatory activities in air quality science and policy making—an equally scandalous case can be made for US EPA work in other areas of responsibility where toxicology and epidemiology are abused and misused to expand the EPA list of targets for regulation and opportunities for EPA to scaremonger.

I also cannot take the time or the space in this discussion to expose the US EPA new area of scientific misconduct and scaremongering—epigenetics and their claims of inheritable acquired toxin carcinogenic genetic mutations—revisiting Lamarck and Lysenko long ago discredited theories about acquired genetic changes. Such irresponsible scares about inherited toxic and cancer effects are ideal for irresponsible aggressive environmental fanatic wannabee regulators and their obedient research army.

US EPA researchers in epigenetics are their new breed of scaremongers with the target people who think exposure to some named toxin might effect their children or grandchildren. The lust of power and influence and cheating on science go hand in hand.

All of what I have exposed above combines to make an effective and urgent argument for the proposed US EPA policy change to promote integrity and transparency of US EPA science in matters of regulatory policy decision making. The time for cleaning up the US EPA scientific perfidy and misconduct, malfeasance is long past overdue.

I anticipate there will be institutions and scientists panicked and anxious about proving up their research assertions and conclusions—a very beneficial and healthy development. The polity will benefit from science and policy making that is based on reliable methods used by researchers with integrity who are subjected to impartial and thorough competent reviews by experts who are not conflicted by ideological, political, monetary or social/professional influences.

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Personal Matters / Ex. 6